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REMARKS ON THE VESTIBULAR TESTS—WITH A RECORD OF THE VESTIBULAR FINDINGS IN A GROUP OF MIXED NEUROLOGICAL CASES.

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The vestibular tests for the localization of cerebral lesions have interested me for some time, so that when the requirement to write a candidate's thesis arose, my thoughts turned to that subject. I thought that testing a group of varied neurological cases, particularly such that are supposed to possess lesions within the central nervous system and probably within the supposed pathways which have been more or less accepted as existing, would make an interesting study to report.

When the work was completed and the startling discoveries that I had hoped to make were not so dazzling, I became even more thoughtful on the subject. It became more vexatious when I recalled that I frequently failed to make neurologic diagnoses with as much assurance as many case reports seem to possess. I therefore set out to make an analysis of as many case reports, syndromes and various statements as I could within the allotted time and determine wherein I failed, or perhaps to find an incomplete justification for diagnoses made from the vestibular tests alone.

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The neurological cases which I have studied, while I could not deduct from them specific syndromes, when summarized, do fit into the general scheme of things. They either yield negative results when according to rules should be otherwise, or at times do manifest signs which in a general way lend themselves to the general classification of possessing characteristics of an intra-cranial lesion. Accordingly I have grouped them and summarized them and placed them at the end of this paper to speak for themselves, either for their negative value or for whatever positive characteristics that they may contain.

Turning to the tests themselves when some of the literature and case reports are studied, one is both amazed and confused to note the wave of enthusiasm expressed for them. The optimism exhibited for their value is tremendous. One feels rather deficient when results of similar certainty cannot be duplicated. It makes one studious, both of one's self as well as of the writings and reports of others. The only consoling feature that I find is that many of my friends and colleagues are as much at a loss as I am. I feel, therefore, justified in putting my thoughts and conclusions into writing after some reading and thinking.

One of the most conspicuous facts in the subject is the ease with which many writers accept the assumed standards of normality as final and the willingness with which they compare their findings as abnormal with those standards. Another noticeable feature is the desire to make neurological diagnoses from the vestibular findings with almost an effort to keep their eyes shut to the neurological data before them; yet these neurological findings seem unconsciously to influence the diagnosis. In some of the case reports and grouped syndromes I have separated the vestibular findings from the neurological reports and try as I may I could not help but think of many patients whom I have examined and who had nothing particularly wrong with them, at least intracranially, and who gave responses similar to many whose diagnoses were intracranial lesions based on those findings. When afterwards the neurological findings in these reported cases were added, a suspicion of an intracranial lesion suddenly crept into being. I concluded, therefore, that in all instances a neurological record is indispensable to making the vestibular findings of value.

Technique and Normal Responses: Most American otologists employ the rotation test, the caloric test and many the galvanic method of eliciting vestibular reactions. These are best described by Jones.[1] Errors in technique are exceedingly possible and one may

find flaws in almost any technique. The simple difference of observing the after turning nystagmus when the patient looks straight ahead as compared to when the patient looks to the side or at a fixed point made a difference in finding the normal nystagmus to last 24 to 40 seconds instead of a few seconds to over a hundred seconds. MacKenzie[2] has thus pointed out an error in Barany's technique in having the patient look to the side which causes a prolonged nystagmus, because the ocular muscle tension caused the increase in the duration of nystagmus and the latter was not due to the pure reflex. Kobrak claims advantages for his method of caloric stimulation. He uses small quantities of water at certain temperatures and obtains reactions by increasing the quantity of water or lowering the temperature. Cevoloto[3] finds advantage in using diathermy to cause stimulation and so gets reactions by varying the amount of current.

In the employment of any technique the nystagmus is more accurately observed than the vertigo and past-pointing, the latter depending much upon the type of individual. Some patients react to stimulation more severely than others even under normal circumstances. One wonders, therefore, while reading case reports how much actually can be deduced from records such as diminished vertigo or diminished past-pointing of a few seconds or inches respectively. I recall reading a case report in which past-pointing of one-half inch is recorded, a space which is more than covered by the average adult pointing finger. Insofar as nystagmus is concerned I can recall any number of patients whose responses were of only 10 or 12 seconds' duration and nothing was wrong with them intracranially or within the labyrinth. The point I wish to make is that the methods at our command are not sufficiently exact in the mechanical sense so as to lend themselves to such fine differentiation as though they were some mechanical instruments of precision. In turning a patient with the head 30° forward much ado is made about the exactness of the degree, yet I doubt if any observer by mere looking can fail to err within 5 or 10 degrees of inclination. There is no provision on any Barany chair that I know of that will gauge this accurately. The same may be said of past-pointing. The slightest deviation of the head by the patient to one side or another would naturally influence the distance of past-pointing and this is bound to happen unless the patient is fixed and strapped in the chair so that he could not possibly change the position of his head. These little apparent inaccuracies, together with reports one reads by scientific investigators which even challenge the physiological facts involved in the tests make one skeptical. For instance, Maxwell,[4] in experimenting

on a dog-fish, lifted the horizontal canal into a vertical position and upon rotation elicited the same nystagmic movements as though the canal were still in the horizontal position. He further expressed the belief that the reactions depended more upon the current in the utricle than that in the canals. Of course, one cannot deny the fact that certain things happen upon the employment of certain forms of stimulation, but whatever they are they must be taken with a great deal of reservation and not as though they were the final thing in accuracy.

When one turns to the interpretation of abnormal reactions the confusion is most appalling. One can find a syndrome for almost any type of case; and even those syndromes which seem to be found most frequently and are generally accepted, find contradiction in case reports with similar lesions which give other syndromes or typical syndromes with strange lesions. To quote two or three instances would be interesting. For instance, the almost generally accepted dictum of cerebellopontile angle lesion—that there is a dead labyrinth on one side with impaired vertical canals on the other side has frequently found exceptions. Linthicum and Rand[5] report a series of cases of concussion of the brain which gave a similar syndrome. Zacks[6] reports such a syndrome in a case of head injury. Cassidy[7] reports a case of cerebellopontile angle lesion with a normal labyrinth on one side and perverted reaction from the opposite vertical canal, otherwise normal. Here was a perverted response but not the classical dead labyrinth. He called neurological signs to his aid in diagnosis.

Conjugate deviation to the side of the lesion on stimulation is found to be a frequent manifestation in cerebral lesion by Fisher and Grant.[8] Keeler[9] reports a case of cerebellopontile angle lesion with conjugate deviation away from the side of the lesion.

The absence of shock reactions in posterior fossa lesions has been looked upon as a certainty, yet isolated case reports show contradiction to this also. Lewy[10] reports such a case.

The reverse is also true, that while great susceptibility to stimulation is supposed to exist in cases of supratentorial lesions, such lesions also exist without susceptibility to stimulation.

As with the clinical observers so it is with the research workers, who record data and statements which contradict fundamentals upon which many manifestations are believed to be based. The centre for the quick component of the nystagmus reaction is thought to be in the cerebrum. Lesions are diagnosed in locations of the brain because of the absence of this component, yet the centre of the

quick component has been placed in different locations by different investigators. Spiegel[11] places it in the nuclei of the pons. Ivy[12] records it as being below the thalamus.

Much is dependent, in diagnosis, on the fact that past-pointing is interfered with in lesions of the cerebellum. These areas involved are referred to as the pointing centres of Barany. Zimmerman and Tcheruchew[13] disclaim the existence of these centres of the cerebellum, but feel that they are found in the pons, medulla and cord. Weisenberg[14] in his studies concludes that there are no centres for deviation in the cerebellum.

This variation of facts certainly gives one food for thought; the least they do is to inspire one with caution. The reported cases and findings with autopsy reports upon which statements are based cannot certainly be denied, but the explanations and interpretations that come with these reports cannot, in view of these variations, be taken as a certainty.

However, that there is clinical value in the manifestations of the vestibular phenomena one must admit in view of the many studies of cases and reports of findings. Their value is not detracted from by the occasional contradictions of isolated cases or contradictory findings of various researchers. These aberrations add to their value as something is learned from all of them.

At the present time the most valuable types of contributions to this subject are the well studied cases with their reports of operative findings or autopsies. Much credit is due to Fisher,[8] Jones,[1] MacKenzie,[2] Lewitt[15] and others who studied their cases and reported the findings in detail. The tendency, however, to group manifestations into symptom complexes, while of great clinical value, would be more so if they were, if possible, supplemented by histological findings in the various parts of the brain beside the gross autopsy reports.

In order to give these vestibular tests the greatest value, there ought to be some way of standardizing reports, methods and interpretations so that all may speak the same language. It is quite common to hear a neurosurgeon speak or see his written statements that they place value in the interpretation of these tests if Dr. So-and-So is reporting. The peculiar thing is that each one recommends his own particular favorite. If the personal element of the examiner has so much to do with the value of the findings, one almost suspects that the favorite's knowledge must be more valuable because of his acquaintance with neurology rather than his knowledge of the vestibular tests. If a certain reaction exists and

means anything, it should mean the same whether Dr. Smith or Dr. Jones found it. Given a tuning fork finding in a deafened patient, if the tuning fork is the same and the method of testing is the same, hardly anyone would question it because one otologist reported on it and not another. Why should the vestibular tests be any different? The possibilities offered by these tests are so great that a committee appointed by some national society to standardize them is at least as justifiable as the committee on tuning forks and hearing tests. The inner ear is the only accessible organ through which the brain can be interrogated and cerebral lesions localized if we could be certain of the anatomic portions involved in each normal and abnormal finding.

Interpretation of Reactions: With the variations of opinions and from things as they exist, one may ask, "How much of value is there in performing the vestibular tests, and what interpretations can be selected as being least misleading if not actually of positive value?" The answer is that everything should be given due consideration and in the present state of our knowledge every reported syndrome or reaction in a given case, while it may be empirical to base any diagnosis on it, eventually they are bound to arrange themselves into symptom groups based upon actual anatomical and histological information.

For the time being a practical working outline, including the most commonly found reactions and syndromes in combination with neurological information, is of value. The use of a uniform chart to make records is essential and tends to standardize methods of testing. The chart presented by Jones,[1] perhaps slightly modified to suit one's needs, is the best yet offered.

The record under the heading of Spontaneous Phenomena has nothing to do with the tests except when the findings are modified by stimulation. The value of the spontaneous findings is proportional to the amount of clinical information brought into play by the examiner or neurologist. These are purely clinical signs. The findings after stimulation, however, require interpretation from the standpoint of the tests and become of greater value when combined with clinical findings of the neurologist.

The spontaneous phenomena which come up for consideration are nystagmus, vertigo, falling and past-pointing.

Nystagmus: Without going into details of definitions and theories of nystagmus, to state briefly, when that symptom is present the

first requirement is to differentiate it from an ocular nystagmus. When the nystagmus is caused by interference anywhere in the vestibular tract from the inner ear to the brain it is always composed of a slow and quick component, is associated with more or less vertigo and ataxia and is increased in intensity when the eyes are turned in the direction of the quick component and decreased when the eyes are turned in the direction of the slow component. When the nystagmus is vertical in direction or oblique, experience has shown the cause to be intracranial. When the duration is only a few days and accompanied by vertigo and is showing a tendency to improve, the lesion is most likely in the inner ear.

Vertigo: There are many causes of vertigo, the one of interest in connection with the vestibular tests being that caused by some intracranial lesion. Usually when all vestibular responses are within the normal range, vertigo is either caused by some extraneous condition or if the cause is intracranial, the tests are unable to locate it definitely by its mere diminution in duration.

Falling is purely a neurological symptom and when it is correctly manifested after stimulation, it merely indicates the presence of vertigo. Little can be learned from it otherwise. Spontaneously, if the falling is uniform, that is, the patient always falls to the same side, it is suggestive of a cerebellar lesion on that side.

Past-pointing: Spontaneous past-pointing of an inch or two or even several inches means nothing except when the vestibular tests are unable to modify it. Then it is significant of some intracranial lesion; whether it is in the cerebellum, as maintained by Barany, or in the pons is still open to question, but experience favors the belief that the anterior part of the cerebellum is involved on the side of the abnormal pointing.

In the interpretation of the reactions to vestibular stimulation, there are many syndromes or group reactions recorded by various observers.

There are certain reactions which experience has shown, with rare exception, to be present only in intracranial disease. These are:

Inverted nystagmus (opposite to normal direction).

Perverted nystagmus (wrong type of nystagmus).

Vertical nystagmus (up or down).

Oblique nystagmus—this type is not a frequent one and a good imagination can delude one very easily. Unless it is observed when the patient's head is in upright position or at least not tilted toward

the shoulder and the eyelids open wide, its presence is often a delusion.

Conjugate deviation of the eyes.

When any of the above reactions are elicited one may be reasonably sure that some intracranial lesion exists.

Next in importance in which the tests are of value is their ability to detect whether or not there is any activity in the labyrinth or eighth nerve, or the activity of any part of the make-up of the labyrinth or nerve. By that I mean the complete absence of the reaction. I have not been able to satisfy myself that a little hyperactivity or even marked diminution in activity is worth any consideration except in combination with neurological data. In the latter instance the diagnosis could almost be made by the neurological findings alone.

Based upon the finding of an inactive labyrinth (including the cochlear portion) is the so-called cerebellopontile angle syndrome—the finding of an inactive labyrinth on the side of the lesion with absent or impaired vertical canal on the opposite side. It is really the non-responsiveness which makes the diagnosis and the abnormal reaction on the opposite side is merely a corroboration of the fact that the lesion is intracranial and not in the dead labyrinth. In the cases that I have seen I always felt more certain if the opposite side manifested a perverted nystagmus or an inverted one. The galvanic current on the inactive side of any part of the vestibular make-up is also suggestive of an intracranial influence—I say influence advisedly, as it may merely mean some extradural irritation.

Past-pointing, unless it is persistently absent in some direction, I do not believe has any definite value. I say this, not unmindful of the fact that diagnoses have been based on its diminution in the number of inches. The variation one sees in the past-pointing from the normal 10 or 12 inches is so frequent in normal individuals that an inch or two or even more hardly seems to deserve any consideration. Its complete absence is a more substantial manifestation and frequently points to an intracranial lesion. The same sentiment can be expressed with reference to vertigo. Its complete absence is significant, but its diminution in time from the accepted standard of 26 seconds is merely problematical.

The above statements, of course, are my personal beliefs based on analysis of case reports and some experience, but as I set out to conclude this paper with a working outline in the examination of patients, its value will be enhanced by briefly listing the more common findings of various observers which they have found helpful.

Many of their conclusions are based on previous experiences and in some instances on assumed intracranial pathways.

The following are brief records:

No susceptibility to stimulation (no shock reactions) suggestive of posterior fossa lesion (8).

Increased susceptibility to stimulation is suggestive of supratentorial lesion (8).

A patient with suspicious neurological signs and has impaired vertigo, past-pointing with poor pelvic girdle reaction and is not upset by the tests—suspicious of cerebellar lesion (8).

A patient with signs of a cerebral lesion when he shows increased susceptibility to stimulation, an exaggerated nystagmus with a corresponding increase in vertigo and past-pointing may signify a lesion near and anterior to the brain stem (8).

Poor pelvic girdle movements are indicative of some brain lesion (16).

Conjugate deviation of eyes point to a cerebral lesion on the side toward which there is deviation (16). It may also be in the decussation of the cerebellar peduncles (1).

Exaggerated past-pointing out of proportion to vertigo with absence of susceptibility to shock suggests a lesion in occipital lobe (16).

Disproportion between nystagmus and vertigo indicates an intracranial lesion (16).

Absence of reactibility to cold caloric in upright and recumbent positions with the continuation of good hearing in a case of leptomeningitis suggests exudate in arachnoid prolongations of 7th and 8th nerves and is of serious prognostic import (17).

Absence of reactibility of the vertical canals of both ears to cold caloric is suggestive of increased intracranial pressure of the posterior fossa (18).

Impaired or absent responses from the vertical canals have been found by different authors to exist in meningitis and temporosphenoidal lobe abscess.

Large excursions as well as small, rapid excursions of nystagmus have been observed in intracranial disease.

All of the above manifestations show much variety and while they are confusing, they have proven sufficiently reliable to warrant

further study. The series of neurological cases that I have examined from the vestibular standpoint follow the same tendency to confusing variations as may be seen from the variety of reactions elicited even in cases of the same pathology.

RECORD OF VESTIBULAR FINDINGS IN VARIOUS NEUROLOGICAL PATIENTS

The following cases were selected from the department of nervous diseases at the Philadelphia General Hospital. The thought in selecting these was to gather a few of each variety and to choose such as showed most pathology. Thanks is due to Dr. N. B. Cohen, the resident on the service at that time, for selecting the cases and gathering the records and opinions of the neurologists and pathologist who had direct charge of these patients. His assistance was most valuable.

The cases chosen include the following diseases: Postencephalitis with Parkinson's syndrome, 8 cases; Parkinson's syndrome, unknown etiology, 1 case; multiple sclerosis, 7 cases; tabes dorsalis, 6 cases; dementia (one of these being acromegalic), 6 cases; pseudo-sclerosis of Westfield, 1 case; lateral sclerosis (cerebrospinal lues), 1 case; Friedreich's ataxia, 1 case. Total, 31 cases.

A complete routine vestibular examination was made in each case; some were repeated to be sure of the correctness of the findings. The idea was to determine what, if any, abnormalities in the reactions were, and if any of them were characteristic in any one disease. As all of the above cases have some lesion or another of the nervous system, the examination promised to be of interest.

For the sake of brevity, only the outstanding abnormal vestibular findings are given, the others being practically within the normal range. A brief summary of the patients' neurological findings is also given so that if any relationship exists between the neurological symptoms and vestibular findings, they can be easily compared.

POST-ENCEPHALITIS WITH PARKINSON'S SYNDROME.

Case 1: I. L., male, age 49 years. *Neurological findings:* Stiffness of right hand and arm with trembling, impairment of speech, reflexes exaggerated, right eye lags in convergence, weakness of right foot with pain, weak motor activity and facial weakness on right side, Wasserman test and spinal fluid negative. *Vestibular findings:* On turning to right, head upright, horizontal nystagmus to left 35 seconds duration and vertigo 38 seconds to left (exaggerated); on turning to left, head upright, vertigo 38 seconds to right (exaggerated), otherwise normal responses.

Case 2: J. S., male, age 43 years. *Neurological findings:* Since 1927 patient noticed difficulty in speech, weakness and tremors of both hands, liquids dribble out of mouth, eyes fixed—staring, seventh nerve—motion lost on both sides, flexed elbows and wrists, staring expression, masked face, superficial reflexes absent, third, fourth and sixth cranial nerves impaired, eyes slow in rolling. *Vestibular findings:* Spontaneous phenomena negative except that the eyes roll sluggishly in all directions. Rotation—With head upright and turned to either side there appeared a mixed horizontal and rotatory nystagmus in the right direction instead of pure horizontal (perverted). There is also a general diminution in responses. The nystagmus upon turning to the right is of 15 seconds duration and turning to the left is of 10 seconds duration. The vertigo on turning to the right is of 9 seconds duration. Turning to the left, no vertigo. There was no past-pointing when turned in either direction. Caloric—All reactions diminished—right side responds in 2 minutes and left side in 1 minute and 40 seconds. All douching was done with cold water, 68° F.

Case 3: B. S., male, age 40 years. *Neurological findings:* Staring expression, pupils irregular and fixed to light, reflexes—sluggish biceps—the triceps, patellar, ankle, and Babinski were all absent; inability to help himself to anything, alternating squint, tremors of the upper extremities, forearms flexed and resting against the chest wall. *Vestibular findings:* Spontaneous phenomena—vertical nystagmus down on looking down, internal rectus paresis right side, otherwise negative; rotation—all responses correct but diminished, e.g., vertigo 8 seconds, past-pointing 6 inches; caloric—all responses correct but diminished—right ear responded in 1 minute and 55 seconds, left ear in 1 minute and 40 seconds.

Case 4: A. S., adult male. No complete neurological record obtainable except that the diagnosis by neurologists was: Post-encephalitis, Parkinson's syndrome with bulbar changes. *Vestibular findings:* Spontaneous—negative. Intention tremors interfered with eliciting past-pointing records. This was also true after stimulation. Rotation—on turning to right, head upright, there was a mixed horizontal and rotatory nystagmus of 25 seconds duration; on turning to left there was the same mixed nystagmus of 24 seconds duration (instead of horizontal—perverted); there was no vertigo reaction. Caloric—nystagmus responses normal—in 30 seconds upon douching the right ear and in 40 seconds when the left ear was douched; there was no vertigo; pointing could not be obtained because of the tremors; the sympathetic reactions of pallor and sweating were present.

Case 5: G. C., adult male. The neurological diagnosis of post-encephalitis was made by the neurological staff, but no detailed records were obtainable. *Vestibular findings:* Practically normal reactions.

Case 6: J. F., adult male. The diagnosis in this case was post-encephalitis. The neurological data was lost. *Vestibular findings:* Spontaneous—negative; rotation—turning to right, head upright, gave normal nystagmus 25 seconds duration, vertigo 18 seconds, no past-pointing; turning to left, head upright, gave normal nystagmus 28 seconds duration, vertigo 18 seconds, past-pointing 4 inches to left; caloric—right ear responds in 1 minute and 29 seconds, left ear in 1 minute and 15 seconds; nystagmus normal, past-pointing normal on right side stimulation and none on left; no nausea, vomiting, pallor or sweating; off and on during the examination the eyes twitched rapidly to and fro.

Case 7: Postencephalitis—mild Parkinson syndrome. *Neurological findings:* Weakness and trembling in right hand and cramp-like pain in right leg and foot, holds arm fixed close to body while walking, tremors accentuated in intention movements, superficial and deep reflexes normal; pain, touch, heat and cold senses normal; hearing impaired in right ear. *Vestibular findings:* Spontaneous—normal; rotation to right and left, head upright, gives normal reaction except duration of nystagmus is increased to 45 and 37 seconds respectively; caloric—right ear responds in 42 seconds; there was no past-pointing and only slight vertigo with head upright or back positions; left ear responds in 53 seconds; nystagmus lasted a little longer than when the right was stimulated; there was an exaggerated past-pointing with the left hand.

Case 8: J. B., male, age 47 years. *Neurological findings:* poor vision, weakness of legs, general retardation of motion; face fixed masklike, eye niche open wide and stares ahead; tremors of eyelids; coarse tremors of the tongue; arms held in semiflexion and does not swing them; tremors of hands and lips; tendency to festination. *Vestibular findings:* Spontaneous—negative except that eyes are fixed and attempted movements sluggish; rotation to right gives a mixed rotatory and horizontal nystagmus, the rest of the responses less active than normal; rotation to left practically normal. Caloric—right ear practically negative except past-pointing is markedly diminished; left ear—responses poor—no past-pointing and no sympathetic reactions.

Case 9: J. T., male age 50 years. Parkinson syndrome etiology unknown. *Neurological findings:* Weakness and tremors of the

hands; weakness of the legs; difficulty in speech and walking—stoops and falls forward. *Vestibular findings:* Practically normal except slight diminution in time of reactions.

SUMMARY OF VESTIBULAR FINDINGS IN THE CASES OF POST-ENCEPHALITIS WITH PARKINSON SYNDROME.

Exaggerated nystagmus when stimulating horizontal canals, 2 cases. Perverted—mixed horizontal and rotatory nystagmus—when stimulating horizontal canals, 3 cases. Marked impairment of vertigo and pointing reactions, 4 cases. Exaggerated past-pointing on douching one ear, 1 case. No past-pointing on douching one ear, 2 cases. Spontaneous vertical nystagmus down on looking down, 2 cases. Normal reaction, 1 case. In a general way most responses were not very active.

Pathology in encephalitis varies. The pathology causing Parkinson's syndrome is probably a peri- and endarteritis, irregular degeneration of posterior columns and numerous amyloid bodies. Changes in the motor cells of the cerebral cortex have been noted.

MULTIPLE SCLEROSIS.

Case 10: H. E., male, age 31 years. *Neurological findings:* Inability to walk, stammering speech, diplopia, headache, nystagmus onset gradual. Pupils react sluggishly to light. Reflexes hyperactive, Romberg positive, ankle clonus positive. Ataxia in all extremities. *Vestibular findings:* Spontaneous—rotatory nystagmus to right on looking to right, to left on looking to the left, vertical upon looking up. Unable to test falling. Rotation—Turning to right, head upright, gave horizontal nystagmus to left 30 seconds duration, vertigo 8 seconds, no past-pointing. Turning to left gave horizontal nystagmus to right 38 seconds, vertigo 11 seconds, past-pointing 2 inches. Caloric—normal but hypoactive responses; past-pointing markedly diminished 2 inches. There was no nausea, vertigo or vomiting.

Case 11: D. S., male, age 67 years. Case of multiple sclerosis with cerebellar ataxia. *Neurological findings:* In 1908 there was difficulty in walking, staggering, indigestion, cramps in the legs, slight oblique nystagmus, tremors of the tongue, positive Romberg. In 1921 there was scanning speech, nystagmus looking to the right, ataxic gait, diminished voluntary motion on right side. In 1925 there was difficulty in speech, nystagmus, blindness in lower half of left eye, adiadokokinesis both hands. In 1930 there was marked

ataxic paraplegia, loss of deep reflexes, diminished vibratory sense, dysmetria on finger to nose test both hands, fibrillary twitching in both hypothenars. *Neurological opinion:* Pyramidal tract involvement with sensory loss, intention tremor, disturbed speech. There is also a cerebellar involvement as well as anterior horn cells. *Vestibular findings at present (1931):* Practically normal reactions.

Case 12: A. H., male, age 53 years. *Neurological findings:* Diagnosis definite, but no detailed data available. *Vestibular findings:* Spontaneous—Staggering, no past-pointing, unable to test falling. Rotation—Turning to right with head upright gave horizontal nystagmus to left 33 seconds duration, past-points with right hand 20 inches, otherwise normal. Turning to left gave horizontal nystagmus to right, past-pointing to left with right hand 20 inches, otherwise normal. The nystagmus in both instances was of rapid movement of the eye balls. Caloric—Responses normal except no past-pointing on douching left ear. Hearing good in both ears.

Case 13: A. A., male, age 49 years. *Neurological findings:* Had nystagmus in 1926, no nystagmus in 1930. Romberg position caused patient to sway; poor coordination. Adiadokokinesis both hands. Spastic gait, stiff legs. Tremors of the eyelids and lower extremities. Eye grounds show angiosclerosis. Difficulty in speech. *Vestibular findings:* Spontaneously—nothing noted. Rotation—Normal nystagmus on turning to right or left. There was reduced past-pointing 4 inches with both hands. No vertigo or sympathetic reactions. Caloric—Right ear no response at all from vertical canals, but poor response from horizontal canal after 3 minutes douching. Left ear same as right. No sympathetic reaction.

Case 14: R. D., male, age 42 years. *Neurological findings:* Illness dates back to 1927, weakness of the legs. In 1930 admitted with disturbance of gait. Tremors of tongue, abdominal and cremasteric reflexes lost. Babinski and increased reflexes on right side, ankle clonus on left. Pain, touch sensation and vibratory sense lost in right leg and up to 1st lumbar vertebra. Thighs show much wasting. *Vestibular findings:* Spontaneous—negative. Rotation—Turning to right gave rapid type of nystagmus of 36 seconds duration. Past-pointing to right 36 inches with right hand and 15 inches with left hand. Turning to left gave rapid type of nystagmus 29 seconds duration. Past-pointing to left 10 inches with right hand and 36 inches with left hand. All responses to rotation were thus exaggerated. Caloric—Practically normal reaction on both sides.

Case 15: J. S., male, age 38 years. *Neurological findings:* Illness

dates back to 1925, when the patient was admitted to the Graduate Hospital. The findings there were: Staggered when he walked, hands shook when he attempted to use them. There were pains in back and the legs, also dizziness and difficult speech. *Vestibular findings at that time were:* Spontaneous—no vertigo or nystagmus. There was past-pointing to left with the left hand 2 inches. Rotation—turning to right there was a horizontal nystagmus to left, large amplitude of 42 seconds duration. Vertigo 16 seconds, past-pointing normal with the right hand but 2 inches in the wrong direction (left) with the left hand. Turning to left gave a tendency to conjugate deviation to the left. Vertigo 16 seconds. Left hand past-points normal and right hand in wrong direction (right) 2 inches. Caloric—Right side gave tendency to conjugate deviation and left hand points to left only and not to right under any circumstances. The diagnosis at that time was given as a possible cerebellar lesion because the left hand never pointed to the right. Also a possible left sided cerebral lesion because of the conjugate deviation to the left. The patient was carried as a case of multiple sclerosis at the Veteran's Bureau from 1925 to 1930. He was admitted to the Philadelphia General Hospital in October, 1930. *Neurological findings at that time were:* Mental function good, staggering gait, falls backward, speech scanning, motor function poor, past-points to left with left hand and to right with right hand. There is pain in the legs. Superficial reflexes normal and deep ones hyperactive. The neurologist's summary and impression: The patient has no loss of sensation, has nystagmus, cerebellar incoordination, no positive signs of pyramidal tract involvement. Impression: Marie's cerebellar ataxia. Lumbar puncture pressure 10 mm., Queckenstedt one side 18 mm., both side 20 mm., quick drop. *Vestibular findings at present, Jan. 17, 1931:* Spontaneous—Slight nystagmus on looking straight ahead, marked horizontal nystagmus to left on looking to the left and marked horizontal nystagmus to right on looking to the right. Past-points 4 inches to right with right hand and 3 inches to left with left hand. Rotation—Turning to right, head upright, gave normal type nystagmus of 40 seconds duration, vertigo 15 seconds to left. Right hand past-points 4 inches to right, but left hand past-points 4 inches to left (inverted). Turning to left gave horizontal nystagmus to right 50 seconds duration, vertigo 17 seconds to right. Past-points 3 inches to right (wrong direction) with right hand and past-points 3 inches to left with left hand (as spontaneous). Caloric—Right ear responds in 30 seconds—nystagmus mixed horizontal and rotary (perverted) to left. Vertigo present, Past-points with right hand 6 inches to right. The left hand does not past-

point. Head forward horizontal nystagmus to right—patient vomited. Left ear responds in 34 seconds—gave mixed horizontal and rotary nystagmus (perverted) to right. There was no past-pointing with right hand and past-points to left with left hand—as spontaneous. Head back gave horizontal nystagmus to right. Head forward gave horizontal and rotatory nystagmus to left—nausea. These findings are much similar to previous ones—six years ago, except that there is no tendency to conjugate deviation. The outstanding feature was the persistent inability for the left hand to point to the right (inward pointing centre impaired), pointing to the left cerebellum as having a possible lesion. Also hyperactive responses and presence of perverted nystagmus from the vertical canals on both sides point to brain stem lesion.

Case 16: H. S., adult male. This patient complained of dizziness and staggering. He died a few days after the vestibular tests were made and an autopsy of the brain showed multiple sclerosis. *Vestibular findings:* Practically normal except that there was no past-pointing upon rotation to either left or right (horizontal canal stimulation).

SUMMARY OF FINDINGS IN MULTIPLE SCLEROSIS.

Spontaneous nystagmus to both sides, 2 cases. Spontaneous vertical nystagmus down, 1 case. Hyperactive nystagmus, 4 cases. Hypoactive vertigo, 1 case. Hypoactive past-pointing, 2 cases. No vertigo, 1 case. Normal responses, 1 case. Hyperactive past-pointing, 3 cases. No past-pointing on one side, 1 case. No past-pointing on both sides, 1 case. No sympathetic reaction, 1 case. No response from vertical canals on both sides, 1 case. Perverted nystagmus, 1 case.

The pathology in multiple sclerosis is the existence of sclerotic patches disseminated throughout the brain and cord. The white matter is usually involved. Of the cranial nerve nuclei the 1st, 2nd and 10th are the most vulnerable. The disease sometimes shows predilection for certain parts of the nervous system, e.g., lateral or posterior columns.

TABES DORSALIS.

Case 17: H. H., male, age 49 years. *Neurological findings:* Tabetic gait, absent knee jerks, positive Romberg, loss of vibratory sensation, pupils irregular and react sluggishly to light. *Vestibular findings:* Practically normal reactions except that the left hand spontaneously past-pointed 3 inches to the left and persisted in doing so after all stimulations.

Case 18: C. B., male, age 49 years. *Neurological findings:* Tabetic gait, pupils fail to react to light, biceps and triceps reflexes absent, impairment of taste. *Vestibular findings:* Practically normal except past-pointing distances averaged 18 inches all the time. Mixed rotatory and horizontal nystagmus on douching the right ear with head upright. Oblique upward to the right nystagmus on douching left ear with head back. There was nausea and vomiting.

Case 19: J. W., male, age 36 years. *Neurological findings:* Tabetic gait, positive Romberg, pupils irregular—left larger. *Vestibular findings:* Normal reactions except vertigo reaction very mild—5 seconds.

Case 20: H. J., adult male. *Neurological findings:* Pupils fixed, nystagmoid movements, patient unable to stand, knee jerk and abdominal reflexes absent. *Vestibular findings:* Practically normal except there was exaggerated past-pointing on turning to right and left, e.g., 34 inches and 36 inches respectively. There was pallor, nausea and sweating.

Case 21: Adult male. Diagnosis tabes dorsalis. Vestibular reactions normal.

Case 22: C. H., male, age 38 years. Diagnosis tabes dorsalis. *Vestibular findings:* Practically normal except slightly hyperactive nystagmus and pointing reactions on turning.

SUMMARY OF VESTIBULAR FINDINGS IN TABES DORSALIS.

Persistent past-pointing of one hand in one direction and no other upon all forms of stimulation, 1 case. Mixed rotatory and horizontal nystagmus on douching right ear, 1 case. Oblique, upward to right nystagmus, head back douching left ear, 1 case. Markedly diminished vertigo from all stimulations, 1 case. Exaggerated past-pointing, 1 case. All normal reactions, 1 case. Hyperactive nystagmus and past-pointing on turning, 1 case.

While the above abnormalities were present in singular instances, the average responses were within the normal range.

Pathology in Tabes: A degeneration of the posterior nerve roots and columns, degeneration of peripheral nerves of special sense; degeneration is upward and may extend to bulbar roots.

MENTAL CASES (DEMENTIA, PARESIS).

Case 23: W. G., male, age 69 years—senile dementia. Patient admitted March 23, 1930, temperature 105, complaining of vertigo

and shakiness. There was blindness in left eye (optic atrophy). *Neurological findings:* Muscle tone normal, absent knee jerks, no ataxia, widened palpebral fissure, convergence absent left eye. On Nov. 15, 1930, had Jacksonian epilepsy on right side. Patient has delusions of persecution. Memory good. *Vestibular findings:* Practically normal reactions except easily made sick by tests. Past-pointing 18 inches when turned in either direction. Douching left ear with head back gives a mixed rotatory and horizontal nystagmus to right (perverted).

Case 24: B. F., female, age 33. Paresis. Patient mentally confused, paretic speech, tremors of tongue, pupils react sluggishly to light and accommodation and are irregular. Blood and spinal Wassermann 4+. *Vestibular findings:* Normal.

Case 25: W. S., male, age 29 years. Dementia precox, Paranoid. Patient has delusions of persecution. *Vestibular findings:* All reactions proportionately diminished. Also does not past-point to right with right hand upon douching right ear and does not past-point to left with left hand upon douching left ear. Both hands fail to point outward upon stimulation.

Case 26: B. Mc., male, age 18 years. Dementia precox—simple type. Patient mentally deficient, reasoning and judgment poor. Hears voices persecuting him. Has superiority complex. Neurologically negative. *Vestibular findings:* Spontaneous—negative. Rotation to right with head upright responses diminished. Rotation to left with head upright no responses. Caloric test gives normal responses for both vertical and horizontal canals.

Case 27: L. B., male, age 27 years. Paresis—also Acromegalic. History of convulsions, mentally retarded. Weakness of left arm, leg and face. Reflexes exaggerated. Pupils irregular—left smaller. Ptosis right upper lid. *Vestibular findings:* Spontaneous—negative. Rotation to right with head upright gives nystagmus 48 seconds duration, otherwise normal. Rotation to left gives nystagmus 44 seconds duration. Caloric—Response in 42 seconds, past-pointing poor.

Case 28: R. B., male, age 26 years. Dementia precox—Hebephrenic type. Patient confused, thinks he is persecuted, has auditory hallucinations—people calling him bad names. Had injury to head during childhood. *Vestibular findings:* Practically normal reactions —perhaps slightly diminished responses. Also upon douching the left ear with head back the horizontal nystagmus changes to an oblique upwards to the right.

SUMMARY OF VESTIBULAR FINDINGS IN MENTAL CASES.

Exaggerated past-pointing on turning, 1 case. Perverted nystagmus upon douching with head back, one case a mixed horizontal and rotatory and one case oblique nystagmus instead of horizontal, 2 cases. Normal reactions, 1 case. Both hands do not point outward on stimulation, 1 case. No response in horizontal canal upon tuning in one side and the other side is diminished, yet the caloric test is normal, 1 case. Hyperactive nystagmus, 1 case.

Pathology in mental cases—functional and no organic lesion discernible.

MISCELLANEOUS CASES.

Case 29: M. K., female, age 36 years. Pseudo-sclerosis of Westfield. Patient in hospital since 1915. Began with shaking and trembling. Gait normal. Head moves backward and forward. Coarse tremors of one arm. At first diagnosed as hysterical tremors. On readmission the tremors were more marked in arm, leg and head. Also there were generalized pains. Speech jerky and tremulous. Lesion suspected to be in lenticular nucleus. Pupils equal and react to light and accommodation. Reflexes present and active. Weakness of lower limbs. Had emotional outbursts. *Vestibular findings:* Practically normal except pointing reactions uncertain because of tremors.

Case 30: H. M., male, age 45 years. Cerebro-spinal lues—lateral sclerosis. *Neurological findings:* Spastic gait, loss of sense of position in both lower extremities. Left hand fingers held in hypertension, right hand shows fingers in flexion. Bilateral Hoffman sign. Bilateral Babinski and ankle clonus. Pupils irregular and react to light and accommodation. Incontinence of feces. In 1921 was struck by automobile and was unconscious for two weeks. In 1925 nystagmus noted on chart. Spinal and blood Wassermann test, 4+. *Vestibular findings:* Spontaneous—negative. Rotation—Turning to right and left with head upright gives exaggerated nystagmus 45 and 35 seconds duration respectively. Vertigo was also markedly exaggerated—duration 45 seconds on each side. Caloric—responses normal.

Case 31: E. G., female, age 65 years. Freidreich's ataxia. Patient in hospital since 1901. Since 1899 she complained of pain in calves of legs caused by flexing. In 1901 patient felt that her knees gave away. She had a brother with a similar condition. Gait ataxic, tremors of body when patient walks, unable to stand with feet to-

gether. Small toes hyperextended, but not the big toes. Reflexes diminished. Nystagmus present. There was lateral movement of the head. Difficulty in speech. In 1905 there appeared definite extension of the big toe. In 1915 the patient had already been confined to wheel chair for two years, unable to walk. Nystagmus upon looking to either side also appeared. In 1925 there was noted ataxic movement of eyeballs, ataxic gait, impairment of posture sense, absent vibratory sense in the legs. *Vestibular findings (1931):* Spontaneous—Rotatory nystagmus to right and left. Rotation and caloric tests practically give normal responses except that there is an absence of past-pointing when left ear is douched with head up as well as back.

Pathology in Friedreich's ataxia shows degeneration of posterior columns, particularly those of Goll, the crossed pyramidal tract, the direct cerebellar tract and that of Gower's. In the columns of Clark are found atrophy of the cells and loss of fibres. Posterior roots and anterior horn cells may be degenerated. The cerebellum frequently is smaller than normal and shows degeneration of dentate nucleus. Atrophy in gyri of the cerebrum has been found as well as changes in the cerebral cortical cells.

General Summary: In a general way the vestibular findings are such as are found in intra-cranial lesions. No definite syndromes characteristic of any one disease could be gathered from these studies. Were the manifestations less conflicting and had we means of eliciting pointing and vertigo reactions less dependent upon the assistance of the patient, possibly each response may lead one to a lesion in a certain part of the brain. Even the mental cases in this series, where no organic lesion is discernible, show some characteristics of those diseases which have a definite brain lesion. With accurate means and perhaps more intimate knowledge of the functions of some silent parts of the brain, definite organic lesions may possibly be traced in these mental cases by these tests.

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THE PALM AND FORK TEST IN THE DIFFERENTIAL DIAGNOSIS BETWEEN CONDUCTIVE AND PERCEPTIVE DEAFNESS, A REVERSAL OF BING'S FINGER AND FORK TEST.*

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Before describing the palm test and discussing its value in the differential diagnosis of deafness, it is appropriate to review the phenomenon of prolonged bone conduction caused by conductive lesions and by artificial obstructions, such as the insertion of a finger in the external meatus, upon which Bing's test is based, and by pressing the palm of the hand against the auricle, upon which this test is based, and compare them with one another.

In unilateral conductive deafness when the handle of a vibrating tuning fork is placed on the vertex of the skull, its sound becomes lateralized in the affected ear (Weber); bone conductivity is increased on the affected side as compared with the unaffected one (Schwabach); and perception of sound by bone conduction exceeds that by air conduction, on the side of the lesion (Rinne negative).

The theories advanced to account for these phenomena have not, so far, met with general acceptance. Bezold, as quoted by Kerrison,[1] believed that as a result of lesions involving the tympanic structures the annular ligament surrounding the stapedian footplate is put under greater tension than normal and as a consequence it impedes the passage of air-borne sound vibrations but favors the transmission of bone-conducted sound waves. The writer,[2] basing his belief on some experiments performed by him and also upon an observation and statement by Gradenigo, as quoted by Dench,[3] to the effect that in some instances of middle ear involvement the auditory nerve responds more easily to the galvanic current than normally, adduced the hypothesis that as a consequence of lesions affecting the tympanic structures, interfering with the transmission of sound by air, there ensues a resultant compensatory hypersensitiveness of Corti's organ, favoring perception of sound by bone.

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When, in normal bilateral hearing, a finger is introduced into the external meatus of one ear and the stem of a vibrating tuning fork is set on the mid-skull, the same group of phenomena will be observed as in conductive lesions, namely: localization of the sound in the obstructed ear (Weber); prolongation of bone transmission on the side of the obstruction as compared with the opposite one (Schwabach); and sound perception by bone will be longer than by air (Rinne).

Under the same conditions as the preceding, when one ear is occluded by pressing the palm of the hand against it, the same set of phenomena will be observed as when it is obstructed by the finger, with the sole exception that all the responses will be greatly intensified.

The explanation of the increased bone conductivity due to the occlusion of the ear canal with the finger is not based, as in the case with conductive deafness, on hypotheses, but is accounted for by reference to a well-known physical principle, as described by Kerrison,[1] and corroborated experimentally by the writer,[4] namely, that the confined air in a closed space will respond with greater intensity, when disturbed by a vibrating body, than an equal volume of air in an open cavity. Since the finger inserted in the meatus converts the ear canal into a closed cavity the air contained therein responds more intensely than in the open canal and since perception of sound by bone conduction is largely determined by the intensity of the disturbance of the air in the canal,[5] it follows that hearing by bone is increased when the meatus is closed.

The prolongation of bone propagation following upon the closure of the canal by pressing the palm of the hand against it likewise depends upon the principle of the closed cavity as against the open canal of the opposite ear, but in addition to this, some new factor, which will be discussed in the course of this article, enters into this process, accounting for its maximum increase in bone conductivity as compared with the former two.

It will be observed, by comparing the prolonged bone transmissibility of sounds in conductive lesions, in finger obstructions and in palm obstructions, that irrespective of the degree of middle ear involvement or manner and degree of obturation of the canal by the finger, that occlusion of the ear with the palm of the hand yields, with few exceptions, the maximum manifestation of prolonged osseous propagation both as to duration and intensity.

Bing's test consists in placing a vibrating tuning fork on the

skull, preferably on the mastoid near the ear to be tested and when the sound dies down a finger is inserted into the ear canal of the tested ear. If the sound again becomes perceptible it is contended by Bing that the condition is due to a perceptive lesion; if the sound is not heard again it is due to a conductive lesion. As it was pointed out by Politzer[6] and by Kerrison,[1] this test is not dependable, for in advanced conditions of tympanic and labyrinthine affections the introduction of a finger into the meatus will not revive the sound and in early or mild conditions of the same affections the sound will be heard again in both conditions on the insertion of a finger in the canal.

The palm test consists in pressing the palm of the hand over the ear to be tested while a vibrating tuning fork is applied to the opposite mastoid. On comparing the prolonged bone conduction produced by tympanic disease, finger and palm obturations, it will be observed that the last named almost invariably exceeds that of the others, both in the duration and intensity, when used according to the method described.

In bilateral normal hearing when a vibrating tuning fork is put on the vertex and one ear is occluded with a finger, the sound will become localized in the obstructed ear. When the sound is no longer heard and the finger is substituted by applying the palm against the auricle, the sound will be distinctly heard again for a variable length of time; if the fork is placed on the mastoid and a finger inserted in the meatus of the same side and then substituted by the palm, as described in the previous experiment, the difference in the results will be more pronounced; and when the fork is placed on one mastoid and the opposite canal is closed with the finger, and when the sound becomes imperceptible it is replaced with the palm, the results are most marked. If a vibrating fork is set on the vertex while one ear canal is closed with a finger and the opposite one is closed with the palm of the hand, the sound will invariably become lateralized on the side closed by the palm. When one ear canal is obturated with a finger and the other closed with the palm and a vibrating fork is applied to the mastoid on the side of the palm, the sound will be heard on the same side; but when the fork is put on the mastoid near the occluding finger, the sound will be heard on the opposite side (in the ear closed by the palm).

In a case of unilateral conductive deafness, when the handle of a vibrating fork is set on the vertex the sound will lateralize faintly or strongly, depending upon the degree of the involvement of the tympanic structures in the deafened ear. Permit the sound to die

out and then apply the palm of the hand against the auricle and the sound will become revived. In the same condition, with the vibrating fork on the mid-skull when the unaffected ear is closed with the palm, the sound will shift its location from the affected to the obstructed ear. In unilateral conductive deafness, if the sound ear is closed with the palm and a vibrating tuning fork is placed over the mastoid of the closed ear, the sound will be heard on the same side, but when the fork is placed on the mastoid, on the side of the lesion, the sound will be heard on the opposite side, the side of occlusion.

In bilateral normal hearing, when both auricles are covered by pressing the palms of the hands against them and a vibrating fork is set on either mastoid, the location of the sound is referred to either the opposite ear or it remains indeterminate.

In otosclerosis, when the pathologic process does not invade the intralabyrinthine structures or cause alterations of tension in the peri and endolymph, thereby producing a shortening of bone conduction, the prolonged bone conductivity is likewise, as in the case of conductive deafness, exceeded by the increased bone transmission resulting from occluding the meatus with the palm.

The explanation for the phenomenon of optimum bone conduction when the fork and palm are used on opposite sides of the skull, as described previously, will become manifest upon a consideration of the factors involved in its production. In a preceding article,[3] certain propositions with their proofs were pointed to as a primary condition to the clear comprehension of the process of hearing by bone conduction in general. They were, briefly stated, as follows:

1. That the outer air (the air surrounding the skull) is set in vibration when a resonating body is brought in intimate contact with it.

2. That the inner air (the air contained in all the cranial cavities) is set in still greater commotion by the vibrating skull.

3. That the air thus set in motion vibrates with greater intensity in a closed space than in an open cavity of the same size.

4. That the intensity of the sound arising from these cavities is in direct ratio to their size.

5. That when a vibrating fork is placed anywhere on the skull, the greatest intensity of its vibrations is produced at a zone approximately opposite to that of the point of contact between the skull and the fork-cross vibration.

Upon considering the occurrence of maximum osseous conduction

resulting from the application of the fork and palm to opposite sides of the skull, it will become apparent that this procedure satisfies the last four of these conditions (propositions). The ear canal is a cranial cavity, hence the air contained therein partakes of the vibrations of the skull. By covering the ear with the palm, the ear canal is converted into a closed space, hence its contained air vibrates more intensely. When the palm covers the ear, not only is the canal turned into a confined cavity, but it becomes considerably enlarged by the inclusions of the spaces found in the inner aspect of the auricle and therefore a greater volume of air is set in vibrations than is possible with the finger in the meatus, hence the greater duration and intensity of the air vibrations in this enlarged cavity as compared with the canal proper. When a vibrating fork is set on one mastoid, the vibrations are most intense at the opposite mastoid, hence the sound is heard best in the opposite ear (the one further from the fork) when it is occluded by the palm.

Since the prolongation of bone conduction produced by artificial obstructions (the finger and the palm) depends upon four of the five conditions (propositions) enumerated above and since that produced by the palm, in the manner described, satisfies all of them, it follows that this procedure furnishes the maximum manifestation of osseous conduction.

It was stated that when both ears are obstructed simultaneously by the palms and a vibrating fork is applied to either mastoid, the sound, as a rule, cannot be lateralized. In this case there are two opposing tendencies brought into play, namely, the tendency of localization (for the sound of the fork to be heard near the point of contact) and the tendency of cross-vibration (for the sound of the fork to be heard diametrically opposite the side of its application). These two tendencies being of about equal force neutralize each other, hence the inability to lateralize the sound.

It has been emphasized in this paper that when the palm is applied against one ear and the vibrating fork is placed on the opposite mastoid, the prolonged bone conduction resulting from this is greater than that arising from any stage of conductive deafness; furthermore, in otosclerosis, without involvement of any of the intralabyrinthine structures, like results are obtained from this test, therefore it follows that when the palm is applied against the ear to be tested and the fork is set on the opposite mastoid and no increase in hearing occurs, the ear is not affected by a conductive lesion nor by otosclerosis. It is precisely on this deduction that the palm and fork test is based, and clinical experience confirms it.

The test is performed as follows: When a patient presents himself complaining of impairment of hearing, a strongly vibrating fork is set on the mastoid of the ear to be tested, or on the opposite one. When the fork sound is no longer heard, instruct the patient to press his palm against the deafened ear or press your own palm against it and apply the fork, which is still vibrating to his opposite mastoid. If the sound becomes revived we may rule out conductive deafness, otosclerosis and early or mild involvement of the auditory nerve. If the sound is not heard again one may conclude that he deals with a marked involvement of the perceptive apparatus or with a mixed lesion of which the nerve element is so predominating that it overcomes the prolonged bone conduction which is an accompaniment of conductive lesions.

The palm and fork test lends itself to objective comparisons.

In normal hearing of both (the examiner and the examined), with a vibrating fork placed on one mastoid of the patient and his opposite ear occluded by the palm, when the examiner will apply his ear in intimate contact with the patient's temporal bone near the occluded ear (on the opposite mastoid from the one in contact with the fork), not only will he hear the sound but he will hear it for about the same length of time as the patient.[5] By referring to the factors responsible for the maximum prolongation of bone conduction under these conditions (the palm and fork at opposite sides of the skull), it will be found that they are alike for the examined and the examiner. These factors, as will be recalled, are: a closed cavity; the increased size of the cavity by the addition of the auricular spaces to that of the ear canal; and the factor of cross-vibration. When the examiner presses his ear against the patient's skull, he not only converts his canal into a closed cavity, but by so doing he likewise adds the inner space of the auricle to it, thus creating an enlarged cavity. At the same time the examiner, by applying his ear to a zone on the patient's skull opposite to the point of contact between the fork and the bone, listens at a point of maximum vibrational activity of the skull. If the patient hears the sound slightly longer than the examiner, it is due either to natural differences in the hearing of both or to the fact that the ear of the patient is in more intimate contact with the vibrating skull than that of the examiner. In this case we have a resonating skull with two hearing apparatuses in contact with it. The patient's ear canal is set in the skull (is surrounded by the vibrating bones), whereas that of the examiner is outside the vibrating skull, but in intimate contact with it. When the examiner, by listening to the sound emanating from the patient's

skull in the manner described hears it considerably longer than could be ascribed to natural differences of hearing, then not only is the diagnosis based on the palm and fork test confirmed, but it becomes a certainty that the patient suffers from an advanced nerve lesion.

In the palm and fork test, as in all functional hearing tests, we must of necessity depend upon the statements of the patient as to the intensity, duration and location of the sound heard by him. Here we are at times confronted with answers that are at variance with the facts. These answers tend to confuse the examiner and induce him to draw erroneous conclusions if he is not on the alert. They are due to the lack of intelligence, co-operation or inability on the part of the patient to properly distinguish and localize sounds especially, if the distinction is a fine one and calls for delicate discrimination. It is the tendency of many patients to refer the sound to the ear nearer the fork or to the site where the fork makes contact with the bone--psychic or imaginary hearing. The errors arising from these sources, in the palm and fork test, can be averted by the following procedure: Apply the palm to the ear to be tested and set the vibrating fork on the opposite mastoid and ask the patient where he hears the sound. If he points to the ear near the fork, remove the palm and wait until he no longer hears the sound, then re-apply the palm and ask him if he hears it again or with the vibrating fork in the same position, alternately open and close the tested ear and ask him if he notices any difference in the intensity of the sound. If his answers to these questions are in the affirmative (and it would be so except in advanced perceptive deafness), then this is positive proof that he actually hears the sound in the opposite ear from the one indicated, since the alternate occlusion and uncovering of the ear covered with the palm would have no effect on the ear near the fork, nor would the covering of the ear with the palm prolong the sound in the uncovered ear.

This test is not intended to replace other well established methods of diagnosis, but rather as corroborative to the others.

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A CASE OF EXTERNAL OTITIS WITH FALSE MEMBRANES, APPARENTLY OF DIPHTHERIAL ORIGIN.

Dr. Philippe Panneton, Montreal, Canada.

Although in the following case the laboratory has not been able either to confirm or to refute clinical diagnosis for reasons that will be given later, the diagnosis seemed to us to be sufficiently probable and the case interesting enough to be reported. External otitis accompanied with diphtheritic false membranes and not involving the middle ear being certainly a clinical rarity.

On March 22 we were called to see Miss S. d. L., age 17 years, for an ear sore. The illness had begun five days before with a feeling of general malaise, headache and slight nasal congestion; all those symptoms being attributed to an attack of "grippe" which was prevalent at that time of the year. But the illness had been progressing in an unusual fashion for "grippe." A strange puffiness appeared in the retromaxillary region. The patient, who had taken to bed, suspecting the trouble to be of dental origin got up and went to the dentist. The latter on the eve of our visit had advised and executed the extraction of three doubtful looking teeth.

For two days, pains had been felt in the auricular region, to which had been added, on the evening previous to our visit, a continuous tinnitus, like the sound of a speeding train. On that day, the ear had started running without any appreciable warning pain and without changing any of the subjective symptoms. The discharge was pink-colored and very profuse.

Personal and family history contributed nothing significant. The patient had had scarlet fever and measles in childhood; general condition was fairly good. There was no sore throat. One brother had had diphtheria in 1918.

At the time of our visit the patient was confined to bed because of fatigue, general discomfort and almost continuous pain, with occasional exacerbation. Temperature, 100.2°; feces, normal. Urine: retention, making the use of catheter necessary. Lungs and heart, normal.

Nose, normal; throat, rather doubtful, but without swelling or actual redness; no pain.

Right ear, normal.

Left ear: Auricle, normal; meatus, discharging a very clear liquid, distinctly pink-colored, so much so that we inquired whether the patient had not had mercurochrome drops in the ear.

Periauricular region: Pretragal gland, as well as retroauricular, could be felt and were sensitive to pressure. Moreover, the whole chain of submastoid glands, including the upper glands of the carotid series and in particular the sub-sternocleidomastoid group were remarkably swollen and painful. Under this swelling one could feel the mastoid apex to be free and insensitive, as was the entire mastoid process.

Auditory canal: This was literally flooded with a pinkish fluid which filled up again as soon as it was drained. Absorbent cotton swabs had to be replaced at least every fifteen minutes, and the pillow was covered with stains. The membranous portion of the canal presented nothing of importance; but towards the bottom one could see a narrowed channel blocked by an opalescent false membrane with clear cut edges, corresponding with the limits of the osseous canal. The impression given was one of a membranous tube introduced into the canal and perfectly molded to it.

The eardrum was covered over with a similar false membrane under which it appeared to be slightly swollen, but without beats. The secretion was flowing continuously from a fissure in the lower part of the false membrane.

Aerial conduction was naturally diminished. There was no history of foreign body nor of scratching. There was no oral pyorrhea.

To sum up, we were certainly confronted with a case of external otitis with false membrane, not involving the middle ear (no tympanic bulging or beats). The fact that the ear duct was involved made us put aside the diagnosis of myringitis and blister of the membranum tympani. It was not a case of parasitic infection. The fever, the secretion, the color of the membrane, the absence of glomerules, the personal cleanliness of the patient, as well as the absence of preliminary itching, in fact everything led us away from the idea of otomycosis.

We had here, without doubt, a case of microbic infection. We knew that Politzer has reported cases of external otitis with false membrane due to various micrococci, and Fraentzel cases due to

Vincent's fusospirillae; that Wilde and others have reported cases of diphtheritic external otitis which Kerrison says are rare, and of which Steinhof has published thirty and Bezold eleven cases.

There happened to be many cases of diphtheria in the district; and that is the diagnosis which we temporarily adopted.

Having expected it to be a common case of otitis media following influenza, we had brought over a paracentesis knife. But unfortunately, there was not at hand the necessary outfit for taking a sample, nor tubes for culture. It was late in the day and there was no danger. We simply prescribed an ice bag, alcohol in the ear every two hours, and then left.

March 22: A visit was paid at noon, this time with tubes for cultures. On this occasion the patient disclosed the fact that vomits were frequent and that there has been difficulty in retaining even liquids. Temperature was down to 99.1° and there was less pain.

Cervical swelling was as before. In the ear, the false membrane was even thicker, now occupying two-thirds of the opening. The secretion had not diminished. The eardrum had changed in appearance somewhat as the false membrane seemed to have a tendency to come away.

We took from the bottom of the canal a piece of membrane which came off easily, leaving a raw surface. The appearance of the duct, that rawness and the persistent glandular swelling decided our action. The patient was given 5,000 units of antidiphtheria serum, and genatropin for her nausea.

March 24: The vomiting had ceased, temperature was the same but the swelling had gone down.

The canal itself had radically changed. The false membrane was only one-third the size of what it was. The pain was much reduced and the general discomfort had completely disappeared.

March 27: The ear had almost cleared, although still running a little. Slight tympanic redness. Ten thousand units were given.

Finally, on April 1, the patient had been out of bed several days and was felling well, although weak. On April 14 she came to my office declaring that she had been quite well for some time. There was, however, to be found, on May 12, a small gland at the mastoid apex, but not painful.

Unfortunately, the culture had given no clue. Examined at St.

Paul's Hospital for Contagious Diseases, it revealed only the presence of staphylococci in small quantity. Upon learning that antiseptics had been used before taking the sample, the bacteriologist informed us that a culture would show nothing and that a negative result would prove nothing one way or the other. In his opinion, the progressive recovery immediately upon serotherapy, together with the clinical aspect of the case, was ample proof to the etiology.

Such is certainly our opinion. We are quite aware that the serum acts sometimes in cases of nondiphtherial infections. But it is a question whether even those cases are not related in some obscure way with diphtheria in masked form. In face of the history of our case, the symptomatology and the results of specific treatment, we feel ourselves justified in making a clinical diagnosis of diphtheria of the auditory canal and believe this to be in all probability diagnostically correct.

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INTRACRANIAL COMPLICATIONS OF OTITIC ORIGIN IN SCLEROTIC MASTOID WITHOUT MIDDLE EAR SUPPURATION. CASE REPORT.*

DR. LOUIS USEN, Brooklyn.

Cases of mastoidal lesions and intracranial complications of otitic origin without suppuration in the tympanic cavity have been reported from time to time. In view of the relative infrequency of such cases, it is the writer's opinion that the case cited below is of sufficient interest to merit description.

Case Report: Colored female, age 26 years, presented herself at the clinic of the Cumberland Hospital on Nov. 11, 1931, complaining of pain of three months duration in the left side of head and behind the left ear. She was admitted to the hospital for observation, where a detailed history revealed the following:

About three months ago, patient had pain in the right ear for one day, after which the pain in that ear disappeared and "settled" in the left ear. From that time on the pain in the left ear became progressively worse and was persistent. In conjunction with this she had tinnitus, felt dizzy and had localized left-sided headache which persisted from the onset until the time she appeared at the clinic. The headache was constant in character, was most intense at night and of such severity that she resorted to aspirin gr. xxx nightly from which she obtained relief for about three hours. She was treated by several physicians who ascribed her ailment to "neuralgia." Throughout her illness she was working full days as houseworker, and at no time during her illness had she been aware of any discharge from the middle ear. She does not remember having had any contagious diseases common to childhood with the exception of tubercular glands of the left neck for which she was operated in 1916, nor could she recall having had running ears at any time.

Physical examination revealed a well nourished female apparently not acutely ill. There was a scar in the left neck, the result of adenectomy in 1916. Lungs, heart and abdomen negative. Nose

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and throat presented no evidence of present or recent pathology. Teeth carious. Right ear drum negative. Left ear drum slightly reddened, no bulging, all landmarks, with exception of light reflex, visible. Hearing markedly reduced. The least pressure over the mastoid and parietal regions caused the patient to shriek and the facial expression registered agonizing pain. A paracentesis was performed and there was no discharge. For the next three days the temperature chart registered excursions from 98 to 103. The patient at no time had actual chills, but complained of "feeling cold." X-ray report read, "on neither side is there the usual arrangement of air cell structure detail. On the right side, close into the base, there is some fine cell air filled structure. On the left side no filled air cell structure detail is defined. There is a general veiling over the region of the base of the mastoids and there is the detail of a fairly large lateral sinus close to the base of the process." The blood count showed R.B.C. 3,200,000, W.B.C. 5,000 with a normal differential, Hemoglobin 45 per cent. At this time (on the third day) a thin, serous discharge was noted from the left ear. In view of all these findings it was decided that we were dealing with an intracranial complication and an operation was decided upon.

Operation: Four days after admission a simple mastoidectomy was performed. Upon removing the cortex great difficulty was encountered due to excessive bleeding, which probably came from the mastoid emissary. The mastoid was found sclerotic throughout and the antrum very small. On reaching the tegmen, thick creamy pus came away under pressure and a necrotic exudate was found close to dura. There was another purulent tract leading to the sinus and upon exposing it, it was found completely eroded by the disease and covered by a grayish, necrotic exudate. The lower end of the sinus was first exposed and then upwards and backwards to within about one inch of the torcula until normal appearing sinus wall came into view. There was no palpable clot present. After being plugged off above and below the sinus was incised. No clot was found. On removal of the upper and lower plugs free bleeding occurred. The plugs were replaced and left in situ. Owing to the poor condition of the patient, it was decided to await the outcome without exposing the internal jugular.

Progress: On the morning following the operation the temperature dropped and stayed within normal limits. For the first time in the past three months patient experienced feeling of well being. During the week, the patient received several blood transfusions. From then on the patient went on improving with the exception of

a persistent low hemoglobin. In view of this fact it was decided to ligate the internal jugular. Consequently this was undertaken. Great difficulty was encountered in exposing the structures owing to scarring of previous adenectomy. In spite of repeated transfusions the hemoglobin remained persistently low, however, ranging between 35 and 45. A search was made for the malarial parasite and none was found. Blood cultures returned negative. Culture from mastoid returned sterile. However, this condition subsequently improved and the patient discharged at the end of the fifth week fully recovered.

DISCUSSION.

In presenting this case several questions arise:

1. Can a lesion, such as described, occur without middle ear involvement, and if so what is the relative frequency?

2. The sclerotic mastoid—wherein does it differ from other types in its pathology and causation of intracranial complications?

3. Is a lesion such as this, associated with a sclerotic mastoid, to be regarded acute or chronic in nature?

4. Is such a manifestation to be regarded as a primary lesion or latent in character?

Of course, it is simple enough to anticipate such lesion in the presence of aural suppuration, but how are we to apprehend such condition in the absence of such findings?

Eisinger[1] states that the findings in the ear drum and the middle ear are not always significant of the condition of the mastoid, since mastoiditis (and its complications) may sometimes scarcely show any pathological changes in the ear drum.

Gruening[2] reported two cases where there was no history of any ear trouble and where suspicion was first directed towards the lateral sinus on account of septic absorption and where an infected thrombus was found in the sinus in each case.

Eagleton[3] states that "although otitic adjacent brain abscess and localized septic meningitis may be present without aural discharge, they are of such rare occurrence that a normal drum membrane with normal hearing practically eliminates a complicating cerebral suppuration." In conjunction with this he does mention the possibility of a mucosus infection, in which the drum membrane is only slightly thickened and lusterless, but in which there is also an associated

deafness, "full feeling" in the ear and tinnitus — of which mention will be made later.

Hvidberg[4] in reviewing the Danish literature finds such cases as a comparatively rare occurrence.

Kerrison[5] states that "while cases have been reported in which the drum membrane has presented no signs of inflammation past or present, there is no evidence in such cases that a tympanic lesion has not preceded and given rise to the mastoid disease."

From the foregoing it may be inferred that an intracranial complication in the absence of middle ear suppuration has at one time or another been preceded by an obscure infection, or the condition may have been latent in character perhaps to be brought into activity through an unknown agency. Kelmer[6] describes a case of mastoiditis and perisinuous abscess which went unrecognized for three months before symptoms referable to the lesion manifested themselves in the form of headache followed by discharge from the ear one week later. The failure for such recognition was due to distraction of attention to severer lesions in other parts of the body. But this does not necessarily have to exist, as in the case under consideration there were no other lesions. He also quotes Gorke and Doderlien, where at autopsy these authors have demonstrated acute otitis media with mastoiditis in patients who had never presented any symptoms whatsoever of such condition during life. According to Krantz,[7] latent mastoiditis occurs more frequently than is commonly believed and that quite often a primary mastoiditis is really latent in character.

We will now consider the next phase. The types of mastoids and pathologic lesions produced by each when invaded by disease. It will not be amiss, therefore, to briefly describe each.

1. *The Pneumatic Mastoid:* In which the mastoid process consists of an outer compact, comparatively dense cortex enclosing within its interior spaces lined with mucous membrane. This type of a mastoid is usually associated with a long cell at the tip where pus collects in the course of a suppurative process.

2. *The Diploic Mastoid:* This is similar to the preceding except that the interior is filled with cancellous tissue. In this type, too, is usually found a large tip cell.

3. *The Sclerotic Mastoid:* In this type the pneumatic and diploic spaces are replaced by compact bone, which in cases of long stand-

ing middle ear suppuration is converted into an ivory-like mass by a process of eburnation with purulent channels here and there.

In the first two, as a result of invasion in the middle ear and its extension into the mastoid process, a coalescent type of mastoiditis results. According to Hanse,[8] such type of mastoid is only associated with an acute and never with a chronic suppuration. According to Wittmaak, any lesion met with a nonpneumatized mastoid process is to be regarded as chronic in nature regardless of the time element.

The difference between a nonpneumatized bone associated with chronic purulency and the one without it, is that in the former sclerosis exists, while in the latter fibrosis results.

However, regardless of the causation of the sclerosis, the lesion produced, insofar as affecting the intracranial structures is concerned, is the same. That is, in contrast to a pneumatized bone in which a coalescent mastoiditis results, the symptoms point first to the mastoid; in the sclerotic type the intracranial complications manifest themselves first. In the case under consideration the cranial aspect of the mastoid did not undergo sclerosis. The collection of pus being under pressure, found outlet in path of lesser resistance to the comparatively thinner roof or tegmen, giving rise to extradural abscess. Similarly, the pus traveling along the pathway between bone and dura was confined to that region, and by pressure necrosis caused softening of and erosion of the sinus wall, providing a path of infection leading to infective sinus phlebitis.

A brief reference will now be made to the bacterial flora in this case. In view of the sterile culture, the probability is that the streptococcus hemolyticus was the activating agent, since this organism is found in about 65 per cent of cases. The streptococcus mucosus is eliminated as a possibility because of absence of its typical manifestations. Also, according to Suepfle, the staphylococcus and pneumococcus rarely give rise to intracranial complications. Kopetzky[9] in speaking of sterile pus says that "the infecting organism plays its part in causing the onset of the lesion, and thereafter the other factors take their share in developing the lesion," and "no bacterial activity is necessary to cause the further unfolding of the pathological picture." Of course, the possibility of error or delay in transmission of the culture tube is not to be overlooked.

An explanation of the negative blood cultures lies in the fact that one does not obtain a positive return in all cases of septic absorption.

According to Libman, negative blood cultures have been obtained in cases of undisputed sinus involvement. An explanation of this is found in the fact that the bacteria may have escaped in the blood stream and have been killed, or there may have been a non-infected clot or an infected clot which did not undergo disintegration. There may also have been secondary foci in the lungs and the bacteria did not find their way into the general circulation.

CONCLUSION.

1. A negative ear drum and middle ear do not always rule out an intracranial lesion.

2. Persistent pain in the ear, localized headache and tinnitus always suggest intracranial pathology regardless of condition of middle ear.

3. Lesions associated with nonpneumatized mastoids are always to be regarded as chronic in nature regardless of the process by which it is brought about and regardless of the time element. Such type of mastoid will tend to produce lesions involving intracranial structures before the bone itself is involved.

4. A mastoidal lesion and its complications may be latent in character for some time, or overlooked because of other lesions in the body, before signs referable to such lesion become manifest.

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PRACTICAL POINTS IN OTOLOGY OF SPECIAL INTEREST TO PEDIATRISTS AND GENERAL INTERNISTS.*

Dr. Wm. Hewson, Philadelphia.

The following notes are meant to correlate a few practical points in otology and it is hoped that they may find some appeal to pediatrists and general internists.

Ear conditions so often explain unexpected temperatures, gastro-intestinal upsets, symptoms referable to the nervous, respiratory and other systems of the body that we no longer consider examination of a child complete without a careful scrutiny of the ears. A boy thirteen years old was brought to me by his mother, who said that he had been coughing for three weeks, since their return from the seashore. The family doctor had examined him and prescribed several cough syrups, etc., without helping the situation. Examination of his nose and throat was negative except for some congestion which might be accounted for by the cough. His ears were also free from inflammation. The drums looked perfectly healthy and he had no wax or inflammation of his external canals. I wiped a good sized grain of sand from the little cul de sac in the vestibule of his right ear and his cough stopped like magic. I do not tell this as a reflection on a fellow practitioner whom I know and admire, but I like to contemplate it as a lesson for any of us. The point is that most of us in examining ears at once introduce a speculum, which rides over this little cul de sac without straightening it and are apt to miss its contents. We are more apt to miss it if we use an otoscope. Let me take this opportunity to advise the more frequent use of a head mirror with reflected light. Otoscopes have become very popular and we are accustoming ourselves to their magnified images, but they give us a restricted field of vision and distort the details. The light spot may be entirely absent with the otoscope and yet normally present with the head mirror. The translucency of the drum membrane may appear very much diminished with the

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otoscope, yet of normal appearance with the head mirror. The external canal wall is almost invariably slighted when examining with the otoscope and in particular that area just within the concha where the ceruminous glands and hairs are most abundant. A good routine to adopt is to examine without the speculum first and then introduce it part way, particularly directing it to the little cul de sac just internal to the posterior cartilaginous boundary of the concha.

The presence of wax in a child's ear is often the real cause for consulting an aurist. By this I mean that pediatrists who find wax interfering with a clear vision of the drum are more inclined to call an aurist than remove it themselves. This is more often than not a wise decision, for in my experience many irritated and inflamed canals are so avoided. Wax should not be a stumbling block if we are properly equipped to deal with it. Very often simply wiping out with a fine cotton tipped applicator will suffice if done before the speculum is introduced. If the speculum is introduced first a partially loose piece may be freed and becomes more difficult to dislodge. Impacted wax should always be removed by syringing and in this procedure let me advise the use of a small syringe with a fine gauge tip. This permits greater force for drilling through the wax and if the stream of water is directed to the margin, it is separated quickly and more apt to come out in one piece. It is rarely necessary to use olive oil, peroxide or bicarbonate of soda to soften wax in children's ears if the above procedure is adopted. Probing and curetting is resented a great deal by children and it is wise to remember that the skin of the inferior wall is thinner and more sensitive than elsewhere in the external canal.

Furuncles are more apt to produce a cellulitis in children's ears than in adults' and the accompanying pain and discharge must not be mistaken for that of an otitis media. The two conditions, on the other hand, may be associated. Furuncles result from external infection penetrating the skin while otitis media is invariably caused by an extending inflammation up the Eustachian tube. Local treatment of an external otitis must be directed only to the affected area or extension and complication will be apt to follow. Ear drops and douching or much manipulation of the ear is contraindicated, for the swelling in the canal may trap liquids and extend the inflammation to the drum or more vital parts. Careful application of tincture of iodine, biniodide of mercury ointment or yellow oxide of mercury and cold or hot packs is all that should be used locally. The general resistance of the patient may be raised by staphylococcic vaccine and other general measures.

Acute otitis media must not be considered as a condition separate and distinct but intimately allied to its progenitors, cold and sore throat. None of us can deny that some degree of inflammation may exist in the middle ear some time before signs of it are manifest symptomatically or through the drum. It is rather remarkable that children's colds do not more often extend into the ears, when we consider how relatively short, direct and patulous are their Eustachian tubes as compared to those of adults. This direct communication is at the same time a factor of safety as well as a source of danger. Colds therefore must be thought of in terms of acute otitis media as otitis media must be thought of in terms of acute cold. Treatment of the one condition must be considered in regard to the influence on the other, being ever mindful of the source of the trouble existing in the nose and throat. I know it is a temptation in treating otitis media to slight the nose and throat, but of the two, the nose and throat should be our principal point of attack. This applies both early and late in the condition.

Early in an acute otitis media the disease is principally confined to the nose and throat, and we should consider means to prevent its spread from this point. One of the factors, little considered, working in our behalf is the sealed off condition of the middle ear cavity. As long as the drum is intact the air pressure and fluid accumulated in the tympanum are continually trying to find exit through the natural channel of the Eustachian tube. The latter through inflammation or congestion is resisting this, but very often if given a chance will give way to the insistent effort of pressure. Treatment of the posterior nares at this juncture helps the situation considerably. The elasticity and distendability of an unopened ear drum is remarkable and should be interpreted as nature's logical effort to establish drainage in the best and most normal way. As soon as incision or rupture of the drum occurs a new order of conditions is established in the middle ear. Protective pressure is removed, unheated and unmoistened air is admitted and infection from the external canal and nasopharynx is almost inevitable. It would be irrational and cruel to insist that every bulging drum be let alone and treatment only directed to the nasal end of the Eustachian tube. The myringotomy knife has given more instant relief from pain than any other cutting edge and the solace to an entire family as well as the little patient cannot be rivaled by any other one procedure.

If an aurist is consulted in a doubtful case, one we will say where the child shows an acute cold, a moderate degree of temperature,

not much earache, no mastoid tenderness, but the drum shows fullness or actual bulging? Do you think his advice would be to incise the drum? It most often would be. The time-honored rule about an ear drum is still good, that when in doubt incise. But if this was your case without an aurist or if it belonged to the aurist without you, would the drum be incised? I venture confidently to say it would not. The difference, of course, is that under constant observation by the one having all the responsibility, such a drum would be given a chance and other methods than myringotomy would be used. If, on the other hand, we have determined to incise the drum we look for a complete evacuation of the infected contents of the middle ear, quick subsidence of the Eustachian inflammation and prompt healing of the drum with a negligible scar and no adhesions. We hope that the incision will entirely relieve the situation, turning the progress of symptoms toward recovery. If we are disappointed one or more of the following conditions has developed: Either

1. Extension of the infection and inflammation to remote or diffuse areas in the middle ear, attic, antrum or mastoid; or

2. Lowered resistance of the patient from disturbed metabolism, endocrine imbalance and general infection, such as tuberculosis, syphilis, measles, scarlet fever and other contagious diseases; or

3. The virulence and selectivity of the infecting organisms is more than ordinary; or

4. Reinfection from the external canal or Eustachian tube.

Considering first the factors favoring extension to the middle ear, we recognize the following:

1. Inflammation of the tubal mucous membrane with congestion, swelling and over-abundance of mucous secretion.

2. Interference with the normal movement of the cilia.

3. Partial absorption of air in the middle ear, which more readily takes place when the mucous membrane is engorged.

4. Gravity, when the head is in the recumbent position.

5. Blowing the nose improperly.

6. Presence of adenoids and obstructing tonsils.

There is no guaranteed method of preventing involvement of the Eustachian tube and extension to the middle ear, but my plea is to

stop neglecting the nose and throat. Simple colds and sore throat undermine our physical structure and it is no longer good practice to consider them casually. The patient's general resistance must be fortified by various means (which you are probably more familiar with than I), but the strictly local seat of these infections must be admitted and dealt with as such. Sinus disease as a cause for middle ear disease is so well recognized that it seems hardly necessary to mention it, but none of us is beyond reproach for minimizing the dangers of a protracted cold, which is in truth a sinus infection.

If our patient can cooperate, a little local treatment to the posterior nares and the Eustachian orifices can be easily accomplished and so the whole tube and middle ear benefited. It is dangerous to use excessive amounts of liquid in the nose in the presence of an early acute inflammation. Reckless douching and spraying should be condemned. Argyrol or other drops which prove irritating should be avoided. Our efforts should be to soothe the nasal mucous membrane and aid the removal of the infected mucous and discharge. Gentle shrinking followed by aspiration and bland oil drops have worked best for me when used once or twice a day.

It has always seemed wrong to me that the sick child with an acute nose and throat infection be placed on its back when the ventral position is so much more conducive to good drainage of the sinuses and ears. I am told pediatrists generally are approving of the ventral position for babies for various reasons and certainly in this particular I cannot urge it too strongly. I have had instances of its benefit in middle ear drainage more numerous than I dare mention and if anyone is skeptical let him try it and see how an early bulging drum may be reduced by this procedure alone.

Blowing the nose is an unstudied art and one that should be universally and properly cultivated. Some children never blow their noses, others blow them too much and most children blow them improperly. Mothers should be instructed to have the child blow with only one nostril stopped, being sure that the nostril which is blown is patulous. Never pinch the nose as is so commonly done at the end of a blow, for it is the back pressure at this time which forces infection into the middle ears and sinuses.

Adenoids are rather commonly understood to obstruct the Eustachian tubes, but tonsils are not often credited with this. It has been my experience to remove tonsils in children whose adenoids were very well taken out, but the middle ear condition persisted until tonsillectomy was accomplished. Anatomical reference backs

me up in the statement that the upper pole of the tonsil frequently impinges on or crowds the membranous portion of the Eustachian tube just external to its pharyngeal orifice. It is not necessarily a question of infection through neighboring lymphatics or the size of the tonsil as it appears through the mouth. The size, shape and consistency of the upper pole of the tonsil are what often count more.

Lowered resistance is so common and often so baffling that a paragraph or two at this time would seem ludicrously brief. I do, however, want to remind myself as well as others interested that cod liver oil and sunlight, artificial or real, should be used as metabolic stimulants in these cases. Whether this therapy affects the endochrines or not is only a conjecture, but small doses of parathyroid, thyroid, iodine and calcium salts should be given to raise the infection resisting qualities of these glands. The acid-alkali balance is a question that concerns us more as the case becomes prolonged. Intercurrent and preceding infection, such as tuberculosis, syphilis and contagious disease should be known of from the first history of the case and when present as a factor should make us particularly guarded in our prognosis.

Local resistance as a separate problem from general resistance is now given more thought than formerly and perhaps explains many of our results from local treatment alone. We are sure that local treatment can often raise local resistance and at times prevent its being lowered. It is therefore reasonable to think of most acute conditions in the ear, nose and throat as beginning with lowered local resistance.

The marked virulence and selectivity of infecting organisms seems particularly manifest during certain epidemics. By observing the character of the discharges and by studying our cases bacteriologically we place ourselves in a better position to use the proper chemotherapeutic measures.

Reinfection of a middle ear after the drum has been opened occurs more often than we like to admit. We can be sure of it when a slight acute discharge, after three or more days, becomes increased and the thin seropurulent character changes to a thick yellowish pus. There are many times, however, when reinfection occurs and the change of character of the discharge is not noted or not demonstrable. The exact source of reinfection in a given case may remain in doubt, but in every acute case with an opened drum we can be sure of two possibilities: first, the external canal through the drum incision, and second, the nose and throat through an unprotected

Eustachian tube. In the first instance the organisms are usually the staphylococci and streptococci, normal inhabitants of the skin of the external canal and though attenuated in violence in their natural surroundings, may develop into voracious fighters in the middle ear. In the second instance the variety of pathogenic organisms is much greater. Various secret sources of infection make their contributions to the unprotected Eustachian tube mouth and it needs no great imagining to appreciate how easily passage is made from there to the middle ear. In acute otitis media with the drum opened, the cilia are impeded by inflammation of the mucous membrane or may be entirely destroyed. The buffer-like resistance of air in the middle ear is also removed as soon as the drum is opened. Air pressure becomes much greater in the nasopharynx than the middle ear during swallowing, coughing, sneezing and certain phases of respiration, and the small lumen of the Eustachian tube acts by capillary attraction to conduct infected material to the middle ear.

No matter from where reinfection is acquired, a different strain of organism is deposited on a mucous membrane which has been prepared for it by the primary infection. Even the air itself admitted through a drum opening may be decidedly irritating because of its unheated and unmoistened state and so predispose to infection. It is conceivable that resistance may be pretty well established to the primary infection, but may be entirely lacking for the secondary invaders. Cultures taken early in an otitis media differ from those taken later and often cultures from an opened mastoid are quite at variance with those taken earlier from the middle ear. Whether the secondary invaders lower the partially established resistance to the primary organism is a question anyone may think about. It would almost seem likely in a case which has developed a surgical mastoid complicating the middle ear infection, when cultures from the mastoid show the primary organism after cultures from the middle ear had shown only the secondary invaders.

Finally, let me urge that after the acute infection in the middle ear has subsided, whether accompanied by suppuration or not, that we direct our patients to have some local treatment to prevent the formation of adhesions in the middle ear. No one can tell whether the thin film of bubbles or sticky threads of dried secretion will persist long enough to act as scaffolds. Fibroblasts may strengthen such most insidiously until later a well marked conductive deafness has resulted which could so easily have been avoided by the proper use of inflation.

6013 Greene and Bryn Mawr Streets.

ATRESIA OF THE CHOANAE: ITS INCIDENCE AND CAUSE.*

DR. J. PARSONS SCHAEFFER, Philadelphia.

Atresia of the choanae and the pharynx may be congenital or acquired, partial or complete. Acquired atresias are more apt to be pharyngeal and congenital atresias choanal in location. The acquired atresias are due to a number of causes: syphilitic ulceration accounting for many of them; surgical trauma and other traumas appear to be the determining factors in some cases; then follow in order, diphtheria, tuberculosis and other simple inflammations. Many of the acquired atresias are, however, not located at the position of the choanae, but lower in the pharynx, really resulting in an occlusion between the nasal pharynx and the oral pharynx.

This paper limits itself to a consideration of congenital atresia of the true choanae. The occlusion may be absolute or partial; the atresic mass epithelial, membranous, osseous or combinations of these. While the condition appears relatively uncommon, its occurrence is doubtless more frequent than is generally believed. In all likelihood some atresias of the posterior nares pass unobserved in the stillborn and in infants that die shortly after birth.

In the living infant with absolute occlusion of the choanae, early signs suggestive of nasal obstruction are manifested. When the occlusion is partial even though the choanal apertures are small, the real condition may not be recognized for some time. Indeed, some individuals go into adult life with partial atresia of the choanae, variously hindering the intake of air by way of the nose. Spontaneous rupture of the atresic mass, especially if thin and membranous, may occur early in infancy. As a rule operative measures are however required to relieve the obstruction.

The explanation of congenital atresia of the true definitive choanae or posterior nares must be sought primarily (1) in the behavior of the primitive choanae, connecting the embryological nasal fossae and

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the early mouth cavity, and (2) the behavior of the floor of the early nasal fossae dorsal to the primitive choanae.

Should the primitive choanae just dorsal to the primitive palate not form during the process of development, the epithelial lining of the primitive nasal fossae and the epithelial lining of the primitive oral cavity would remain intact, each separated from the other by the thinned-out mesenchymal floor of the early nasal fossae. The continued dorsal growth of the nasal fossae into the mesenchymal mass of the nasofrontal process would ultimately, in cases of maximal growth, lead to a condition in which the epithelium of the nasal fossae would come in contact with the epithelium of the foregut, i.e., the nasal portion of the pharynx, with a variable amount of mesenchymal tissue between the epithelial surfaces. This would result in blindly-ending, definitive nasal fossae dorsally; in other words, an atresia of the choanae or posterior nares. If the amount of mesenchyme between the pharyngeal and nasal epithelia should remain thick, an osseous atresia would ultimately be formed. The atresia of the choanae would therefore be membranous, osseous or a combination of these, depending on the degree of dorsal growth of the nasal fossae and the extent of the resorption of the mesenchyme between the nasal and the pharyngeal epithelia.

Even though the primitive choanae are established normally in the roof of the mouth, should the floor of the primitive nasal fossae dorsal to the primitive choanae not be resorbed, there could be no connection with the nasopharynx save by way of the mouth and the oral pharynx.

The palatal processes, developing beneath the floor of the early nasal fossae and fusing with each other in the mid-line in the formation of the secondary palate, would come into intimate relationship with the persistent primitive floor of the nasal fossae dorsal to the primitive choanae and with the nasal floor when the primitive choanae are not established. Fusion between the palatal processes and the primitive nasal floor would in time take place.

307 S. Eleventh Street.

NASAL SINUSES; PRACTICAL CONSIDERATIONS OF GENERAL INTEREST.*

DR. HENRY M. GOODYEAR, Cincinnati.

I shall endeavor to present in a general way, inserting a few brief case reports, our present status in regard to nasal sinuses, and conditions pertaining to them which I think might be of interest to men in medicine in general. Not all the sinuses are present at birth, only the ethmoid cells, a small antrum, and usually a small bony cell at the sphenoid. At the age of three the sphenoid is well founded, and at ten years it has, first of all the sinuses, reached its adult size.

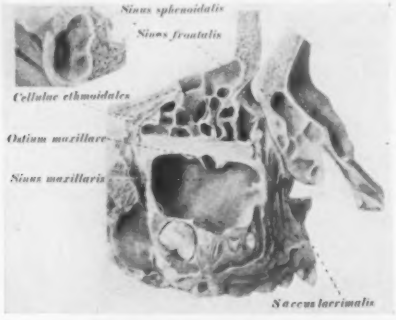

Fig. 1. Note the well developed ethmoid labyrinth at the age of 6 years.

The ethmoid cells, both the anterior and posterior groups, grow rapidly[1] (see Fig. 1) and usually reach their full size by the fifteenth year, and early chronic infections of these sinuses may well be established long before this time.

The frontal sinuses, which represent an upward growth of the anterior ethmoid cells, appear at the second and third year but are not apt to be of clinical significance before the eighth or tenth year of life, when they have reached a fair degree in size (see Fig. 2). Their development continues, however, to the eighteenth and even

*Read before the Cincinnati Academy of Medicine, Oct. 10, 1932.

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twenty-fifth year of life before reaching the full adult stage. Failure of the anterior ethmoid cells to develop, as might occur in early infections, may mean the absence of one or both frontal sinuses.

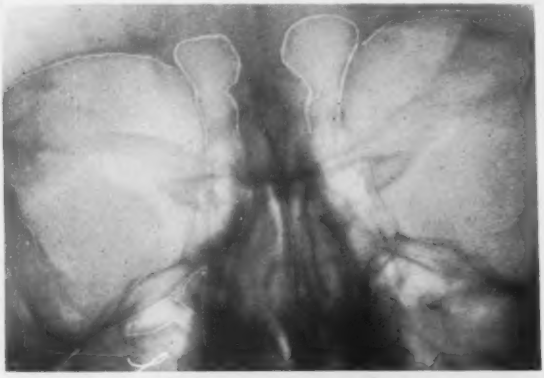

Fig. 2. Frontal sinuses at the age of 10 years.

The maxillary sinuses are the most constant of all, being rarely absent. During the first year of life they are about the size of a pea, distinctly flattened, lying medial to the orbit and not extending below the line of the orbital floor[1] (see Fig. 3). The growth is in a downward direction, and until the teeth erupt there is little room for these sinuses.

Fig. 3. Photograph of a semifrontal section of a child's face, age from 16 to 18 months. Note the normal medial position of the antrum to the orbit and its relation to the inferior meatus at this age.

During early childhood the antrum is so narrow that an instrument inserted into this cavity may readily be passed through both the medial and lateral walls into the soft tissues of the cheek. Under six or eight years of age it is safer, and usually easier, to irrigate through the middle meatus. I have been rather surprised that the normal ostium in these cases is rather larger and easier to enter than that of most adults. I have had a number of children six to eight years of age whose antra I have irrigated, with a double curved cannula, through the normal ostium, under local anesthesia, without great difficulty.

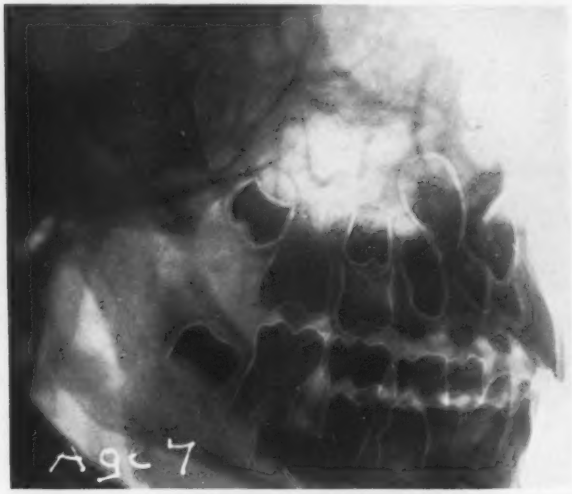

Fig. 4. Note relation of deciduous teeth to the area of the canine fossae. Age 7 years.

To attempt a radical operation through the canine fossae before the permanent teeth have erupted is impossible without danger of injury or destruction of the deciduous teeth (see Fig. 4).

Infection of the nasal accessory sinuses occurs much more frequently in young children than is usually suspected. The membrane of the nose is continuous with that of the sinuses, and it stands to reason that marked swelling of the mucous membrane of the nose must be accomplished during each attack of coryza by a degree of corresponding changes in the lining of the sinuses.

These changes may constitute the early red swelling of an acute inflammation, or the change may advance to that of marked migration of leukocytes with heavy mucus and pus in the sinuses; usually the ethmoid and maxillary. In the majority of these cases the sinuses drain normally and the patient recovers from his attack of coryza (with pus) in seven to fourteen days.

Any condition in infants and young children that might arise from a focus of infection may result from sinus disease. Most common among these conditions may be mentioned: bronchitis, bronchiectasis, asthma, pyelitis, nephritis, rheumatic fever, malnutrition, numerous ocular conditions and recurrent fever in infants and young children without apparent cause.

Several years ago a boy, age 7 years, came to my office with a history of nervous attacks at night that were gravely maniacal in character. The child would cry out, spring from his bed and amid terrifying yells attempt to get out of the window. It was with difficulty that he was restrained and quieted. Both antra were found to be infected. An intranasal antrum window was made on each side and the recovery was immediate. Three years later there had been no recurrence and the child was apparently normal.

Not many years ago most physicians felt that children did not get sinus infections very often, since they did not complain of headache and other symptoms were not regarded. Perhaps one of the pictures most frequently seen in children suggesting a chronic infection of the sinuses is that of the underweight, listless, pale child with nasal discharge, difficulty in nasal breathing, often cervical adenitis, and the history of frequent prolonged colds. Increased lymphoid tissue on the pharyngeal wall is also suggestive.

In the treatment of these established cases, if large tonsils and adenoids are present, and the general condition of the child will permit, the tonsils and adenoids should be removed immediately to give increased aeration and drainage. A large adenoid alone may, by its size, retain a cesspool of mucopus high in the nasopharynx. The maxillary sinuses, and even the sphenoids, may be aspirated and irrigated if necessary.

It is important that we do not overlook an allergic condition which produces an edema of the tissues and paves the way for a superimposed infection. Allergic patients have great difficulty in nasal breathing, frequently sneezing, "itching nose" with watery discharge. The eyes are also often irritated. These patients present a wet, pale

gray, swollen nasal membrane suggesting an alkalosis rather than a tendency to acid. I have been impressed by the frequent history of hay fever, asthma, urticaria, migraine or eczema in the antecedents of these patients. I feel sure that this relation is present in well over 50 per cent of the cases.

Of foods the most common offenders are milk, eggs and wheat, while among foreign contacts other than the grasses and ragweeds, cats and feathers have come most frequently to my attention.

Outside of specific food elimination, or desensitizations, calcium gluconate has been most helpful. In the past few months, however, I have had some rather striking results from the use of dilute nitrohydrochloric acid. Two cases this past summer, both of whom had no results from injection for ragweed a year ago, had perfect relief this summer by taking dilute nitrohydrochloric acid.

This procedure is just the opposite of our previous plan to alkalinizing allergic and hay fever patients. I believe the discovery of this fallacy constitutes a late and very important contribution to medicine. Recently I have seen most astonishing results by the use of acid in several severe, persistent perennial hay fever cases. I have also noticed for some years that a number of my patients have obtained much more relief during hay fever from an acid rather than an alkaline nasal spray.

Experimental work along this line will be greatly extended during the next year. Many cases of perennial hay fever, and apparently many annual cases as well, are in a state of alkalosis, which is only aggravated by further alkaline medication.

There is no doubt that a number of children with much nasal discharge, frequent colds, and a tendency to sinus infections, are low in thyroid. I shall sight as an example a boy of 11 years of age, who came to me eight months ago with profuse nasal discharge and constant heavy mucopus on the floor of the nose. Local treatments, maxillary sinus irrigations and iodine internally did not give any noticeable relief. One-half grain of thyroid was given twice a day with almost astonishing improvement of the nasal condition. I believe we overlook many children with a mild hypothyroidism. This, I think, is especially true in children with a beginning atrophic rhinitis. An early atrophy is often as important a symptom of hypothyroidism as is a dry skin.

In adults infection of the ethmoid and maxillary sinuses predominate.

We have been taught for many years that infections in the upper sinuses gravitate to the maxillary sinuses, but I have been able to demonstrate[2, 3] that iodized oil placed in a maxillary sinus will, with the head in the nose-forehead position, drain into the ethmoid and frontal sinus during the short time required to make a Roentgen exposure (see Figs. 5 and 6). With these pictures in mind you may judge how quickly an ethmoid or frontal sinus may be infected by pus from the antrum when the patient lies down at night.

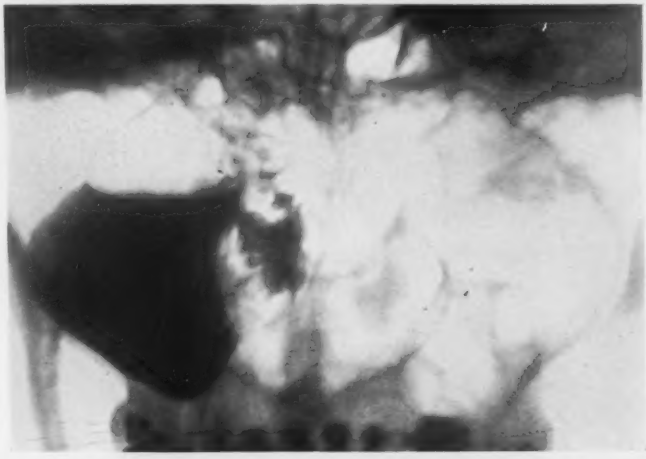

Fig. 5. The Roentgenogram was taken in the nose-chin position. Antrum shows a normal filling. To be compared with Fig. 6.

Often with an acute antrum infection with pain or tenderness over the cheek, and a sensation of the upper teeth being too long, there is frequently an supraorbital pain, medially, which may lead us to overlook the maxillary sinus. I have also seen rather severe occipital and temporal headaches relieved by a radical operation on this sinus.

Irrigations of the sinuses are contraindicated in the very acute stage of infection. If they are necessary it is better to wait four or five days, or even a week, after the onset of an acute infection. After the irrigations are started I believe they need not be repeated oftener than at two or three day-intervals. We do occasionally see a patient who reports having had antrum lavages daily for a month or more.

Very often if the treatment is stopped for ten days and the antrum given an opportunity to dry out the patient is well. All abscessed and devitalized upper teeth should be removed in any maxillary sinus infection.

If, after a fair trial of two or three weeks with irrigations, suction and local medication, there is no relief, an intranasal opening into the antrum should be made to provide aeration and drainage and easy access for further medication. The turbinates are not impaired

Fig. 6. Same patient as in Fig. 5, approximately two minutes later in nose-forehead position. Note the rapid migration of oil to the frontal sinuses between exposures. Pus in the antrum must extend rapidly toward the ethmoid and frontal sinuses, especially while the patient is lying down at night.

in this operation. With our present instruments, and with the inferior turbinate displaced upward it is possible to curette a large part of the antrum wall through a window resection.

On several occasions I have advised patients with very bad antra to have the operation done through the canine fossae (radical antrum), but on their refusal I made a large opening intranasally and curetted the walls of the sinus as best I could and, to my surprise, the results in these cases were most gratifying. One case I remember particularly was a man, age 58 years, with a long-standing infection secondary to an abscessed tooth which had been removed. The pus was constantly foul-smelling after prolonged treatment. Two months after an intranasal operation the patient had gained

fifteen pounds in weight, and the antrum was free of secretion. Three years later I examined this man. He had experienced no further sinus trouble and the lining of the antrum appeared to be smooth and perfectly healthy.

I believe we do many radical antrum operations on patients who would be equally relieved by a more conservative intranasal operation, thus avoiding a disturbance of the nerves to the upper teeth, which always occurs to some extent in all commonly called radical operations.

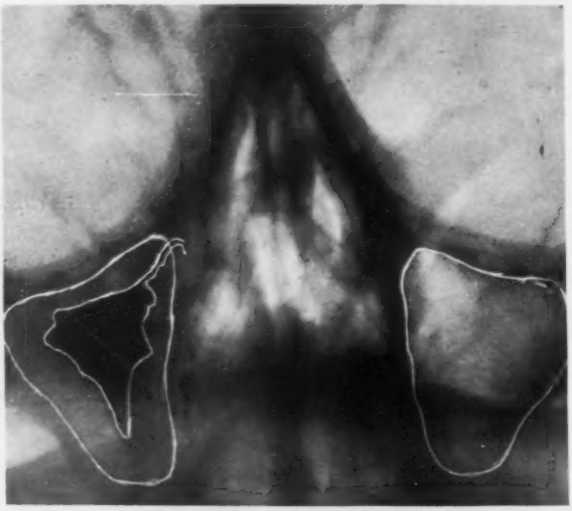

Fig. 7. Man, age 32 years. Bilateral retrobulbar neuritis. Seven teeth removed, tonsillectomy, submucous resection, and both sphenoids opened without relief. Roentgenograms showed slight grayness of right antrum, lavages were negative. Opaque oil showed above filling defect. Radical antrum operation was followed by rather spectacular return of vision, right, 20/20; left, 20/30, with small central scotoma.

You may ask what happens after the diseased membrane is completely removed from a sinus. Does another normal membrane regenerate? In a healed sinus after operation the scar tissue is covered by a new ciliated epithelium.[4]

Nine out of ten adults selected at random will, I believe, show some haziness of one sinus or another on Roentgen examination. In the past many patients have been operated on because of this slight thickening of the sinus membrane; a thickening which may occur

and be permanent after any prolonged, severe attack of coryza. For these patients it would have been better had there been no X-ray at all.

In the past five years opaque oils (iodized oils) have come to be widely used in the diagnosis of obscure nasal sinus conditions. Many

Fig. 8. Membrane completely removed after Roentgenogram (Fig. 7).

Fig. 9. Typical orbital cellulitis with abscess, which was drained with excellent recovery.

important cases show a haziness in Roentgenograms and a negative lavage, yet with opaque oil a definite filling defect is found. It is these patients especially, suffering from some undetermined source of infection, that may be relieved by operation or saved from an unnecessary exploration of the sinuses (see Figs. 7 and 8).

We know now that many suspected tuberculous infections of the chest are nontuberculous and secondary to a chronic sinus. Mullin[5]

reports that cough is present in about 20 per cent of all patients with chronic sinus infections.

Intraorbital cellulitis or abscess arising from infection of the sinuses is always a serious thing (see Fig. 9). I have seen a number of these cases in the clinics and in my practice and I have come to treat them more conservatively. I have found that you can usually wait several days without increased danger to the patient. During that time the infection is being walled off. If a flow of pus can be started from the nose the recovery is usually good without an opera-

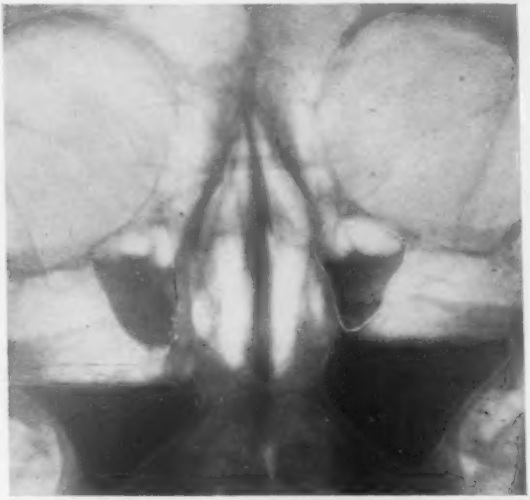

Fig. 10. Opaque oil in antra and infraorbital ethmoid cells. In any radical antrum operation these cells must be opened and the operation extended on into the ethmoid labyrinth if necessary.

tion. As long as the eye is still movable I am inclined to wait before operating. Then only sufficient surgery should be done to provide drainage; an external incision and, perhaps, the opening of the anterior ethmoid cells. If the recovery is not complete, radical sinus surgery can be done more safely later.

Teeth may cause many antrum infections. At their extraction an opening may occur into the antrum. The dentist is tempted to irrigate and treat through this dental opening, as did Highmore, who

first described the antrum in detail in 1651. From repeated experiences I am convinced that this method is wrong, and many active, chronically infected antra result from this procedure.[6]

It would be better to suture the opening immediately to prevent further mouth infection, or to let a good blood clot remain as a base for healing. Even *with most liberal consideration* the canine route should not be used for drainage for more than a few days or a week. If pus arises in the antrum it may be removed by irrigation through the nose. In persistent, more chronic cases with granulations on the floor of the antrum, a window should be made and the floor curetted, and the sinus packed with iodoform gauze.

No one should do a radical maxillary sinus operation unless he is prepared to extend the operation into the ethmoid labyrinth (see Fig. 10), and even the frontal if necessary.

Fig. 11. Patient, age 29 years, had intranasal frontal sinus operation; the opening was good but the headaches continued. Injection of opaque oil showed the intranasal opening had been made into a large supraorbital cell and the main cavity of the true frontal sinus had not been entered.

In exenterating the ethmoid cells the middle turbinate should be preserved unless it is badly degenerated.

To date, no external frontal sinus operation has been devised that does not fail in many cases. Of equal importance with the operation itself is a prolonged and careful after-treatment to insure a free,

permanent opening into the nose. Unless this is maintained the operation is a failure. No matter how large the opening is made, or the type of operation done, the nasofrontal duct will close in certain cases if left alone to heal, without frequent and prolonged dilatations (see Fig. 11).

In a large per cent of cases where the anterior ethmoid cells are well developed and the tendency to a sclerosis is not marked, an intranasal frontal sinus operation without external scarring gives relief from headaches and provides a permanent opening.

Most sinuses would remain free from chronic infection if proper aeration and drainage were always possible through the normal air passages. Such an ideal breathing space is most frequently prevented, outside of allergic tendencies, by the nasal septum, which is rarely without deflection and often presents spurs, ridges and deviations which bring adjacent mucous membranes in contact. Such contact means chronic irritation, and pressure on the middle turbinates may be so great as to completely obstruct the openings of the ethmoid, frontal and the maxillary sinuses.

It is evident that, with such a condition present, with each succeeding attack of coryza there will be a more prolonged recovery and permanent pathological changes will occur in the sinuses, often accompanied by headache and asthenopia.

A submucous resection of the nasal septum is the most technical, yet the most important, and the most uniformly satisfactory of all nasal operations. It frequently fails, however, because it is taken too lightly and deflections are left high in the important area of the middle meatus and middle turbinates, or ridges and spurs are not completely removed.

If obstructions are corrected and normal breathing space established between the ages of 14 and 20 years, there would be very few sinus operations later in life, save for those infected by devitalized teeth. Even at this early age many ethmoid sinuses are already showing hyperplastic changes, but they should not be opened at this time. With the obstruction removed nine out of ten of these cases will recover in one or two years and be far less troublesome to the patient than an ethmoid labyrinth opened and distorted early in life.

Do a submucous resection, retain the turbinates, and give the ethmoids at least a fighting chance to recover. If time proves that the

infection has advanced too far for a nonsurgical recovery, an ethmoid exenteration can be done with equally satisfactory results.

Acute sphenoid infections are often accompanied by rather severe pain at the root of the nose, the temporal and occipital regions, and over the vertex. The temperature may be low-grade and rather continuous, resembling typhoid, as it did in the following case: A girl, age 17 years, who was entered on my service at Cook County Hospital, gave the general impression of a typhoid. Careful physical and clinical examinations, including a special examination of the nose, did not reveal the true cause of illness. At the end of one week the patient suddenly developed meningitis, and at autopsy a closed sphenoid was found filled with pus. A number of cases resembling this one have been reported, with similar findings at autopsy.

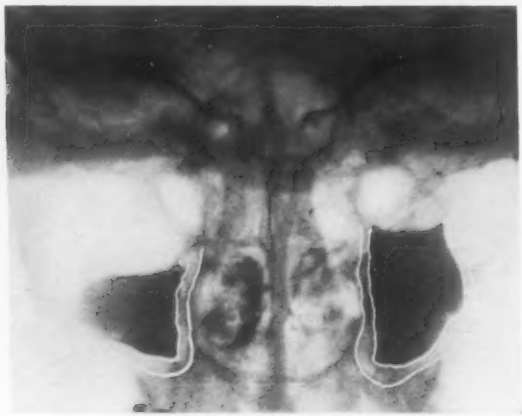

Fig. 12. Patient, age 48 years. Definite history of allergy with eosinophils in nasal secretion. Note the even filling defect bilateral which is rather characteristic of an allergic edema. Two weeks later, this edema had disappeared.

Sphenoids and posterior ethmoid cells causing a sudden blindness (central scotoma) rarely if ever contain any visible pus. Often there is a history of a mild coryza a week or ten days previous, and the blindness is usually first noted on awakening in the morning.

The pendulum is swinging toward more conservative surgery of the nasal sinuses, with greater and more careful attention to establishing a normal breathing space without the removal of vital structures.

Perennial types of hay fever, hyperthyroidism, food disturbances, and general constitutional conditions are receiving more consideration than ever before in the treatment of nasal sinus conditions (see Fig. 12).

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556 Doctors Building.

REPORT OF A CASE OF ADENOMA OF THE PITUITARY, PRODUCING HEADACHE OF A TYPE CAUSED BY SINUS DISEASE.*

DR. HERMAN B. COHEN, Philadelphia.

My purpose in reporting this case is for academic interest and to emphasize the importance of thorough history taking and study, irrespective of the chief complaint. As to the gross and microscopic diagnosis in this case, it is irrelevant for the moment, as the growth was not removed and may not require surgical intervention. Every patient that visits us, complaining of temporal and vertical headache does not have to have ethmoid disease, nor does the patient complaining of occipital or postaural pain and headache have sphenoid disease. This error has been made in the case here reported, where intranasal surgery brought no relief.

History: Mrs. F. S., age 33 years, a white Jewish housewife, was first seen by me, November, 1930. The chief complaint was headache, of varying intensity, dating back seven or eight years; at times occipital or temporal, or both; vertical occasionally; associated at times with nausea and lacrimation. Frequently there was a sense or feeling that there was "something trying to force her head open," a symptom, incidentally, often complained of in acute empyema of the sphenoid. Other symptoms were pain in the right ear and occasionally nasal stuffiness. No nasal discharge, but she did have some postnasal discharge. With a diagnosis of posterior sinus disease, she had been operated upon intranasally, with no relief. Since then there has been some impairment of hearing in the left ear with occasional vertigo, and pain in the right ear continued.

The past medical history is unimportant with the exception of a shock received two or three years after her marriage, following which her menstrual cycle ceased. For this she was operated upon but no cause for the amenorrhea was found. One ovary was removed. This was followed by an increase of thirty pounds in weight. The G. I.

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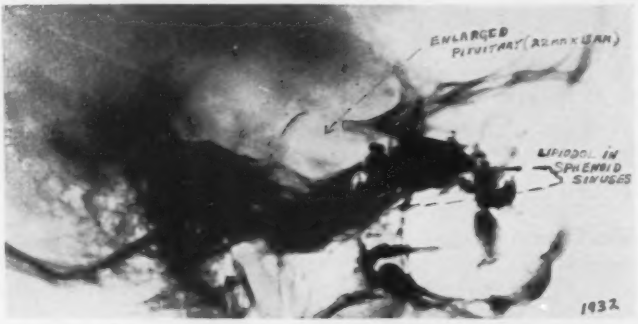
and the G. U. tracts were normal and there were no serious illnesses. The patient did not conceive during the early married period. Under diet and other care her weight was brought down to 130 pounds. The patient is five feet two inches tall and the features are small and chubby. There is nothing acromegalic in appearance.

The eye grounds were normal and on several examinations the visual fields were questionably negative. The B. M. R. was minus five and other laboratory studies were negative. By transillumination the writer found questionable ethmoid disease but the rest of the examination was negative and irrelevant to repeat here.



During one of the visits, the patient stated that she felt that all of her trouble was in the nose, that there was more postnasal discharge and the headaches were quite unbearable. She expressed the hope that something would be done. I discouraged any surgical interference for the time being, but compromised on an X-ray study with lipiodol injection. The study made by Dr. J. Gershon Cohen, I believe, clinches the cause of the headache and from the duration and lack of more serious symptoms and findings, the diagnosis of adenoma of the pituitary was made by him. The report follows: "Omitting detail, there is thickening of the mucous membrane but slightly of the frontals; some slight hyperplasia of the posterio-ethmoid cells and sphenoid. Completely overshadowing the changes of the mucous membranes is the presence of a moderate enlargement of the sella turcica, which measures AP 22 mm., depth 13 mm. The dorsum sella is considerably atrophied and slightly dislocated posteriorly. The posterior clinoid processes are very thin and there

is some erosion of the anterior clinoids. There is a loss and dissolution of irregular portions of the bony confines of the sella floor anteriorly. There is probably, however, no communication between the sella turcica and the underlying sphenoid sinus, as determined by the lipiodol. These Roentgen findings would indicate the presence of a pituitary tumor, intrasellar, probably adenomatous."

The patient under X-ray treatment and checked by B. M. R. test and blood counts has improved considerably and may escape surgery.

It is therefore well to remember:

1. That all headaches and pains in various parts of the head, corresponding to sinus headache areas, are not due to sinus disease, and that a corollary would be.

2. That affections and diseases of distant and of organs proximal to the sinuses may also produce headache and other otorhinologic symptoms besides sinus disease *per se*.

My last progress note relative to this patient is of Oct. 31, when her statement was: "My headaches are about 95 per cent better; I have no nausea or watering of the eyes."

The Lenox.

POSTOPERATIVE PULMONARY COMPLICATIONS; ARE THEY PREVENTABLE?

DR. CHEVALIER JACKSON, Philadelphia.

This subject may be considered best by presenting clinical cases arranged in three groups: *1.* postoperative atelectasis; *2.* posttonsillectomic abscess; and *3.* cases of inspiration of instruments and other substances at the time of operation. These three groups are quite distinct. Postoperative atelectasis follows abdominal and occasionally pelvic operations; it is not primarily suppurative, and we do not see it after tonsillectomy or other operations about the head or upper air passages. In Group 2, on the contrary, a sudden onset with fulminating symptoms of abscess, including foul expectoration, follows immediately or shortly after a tonsillectomy or an operation about the upper air passages. This type of suppuration does not follow abdominal or pelvic operations unless due to septic infarct. In Group 3 we have the long symptomless interval characteristic of foreign body inspiration. I have taken out in ten cases portions of tonsil instruments, in sixty cases teeth dislodged at operation, and in one case a tonsil. In all of these cases there was a symptomless interval, and no suppuration except in those cases in which the foreign body had been present in the lung for a long time, months rather than days. In the case of the tonsil, the tonsillar tissue, containing presumably all infections usually present in diseased lymphoid tissue, was in the bronchus seven months without producing abscess; for the first three months there were no symptoms. It is obvious that the whole question of postoperative suppuration is closely related to the whole problem of descending infections from the upper air passages to the lungs. Until we have determined the route of infection and the other etiologic factors, it will be impossible to answer the question as to whether or not these complications are preventable. The route by the air passages is very direct, as is proven by the foreign bodies lost down at operation; the route by the blood vessels is equally direct, as shown by Fetterolf and Fox. The route by the

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lymph channels may be from the field of operation in the upper air passages, or from the tracheobronchial tree by way of the local lymphatics in the lung, though many questions in regard to the latter group of lymphatics are as yet unanswered. There can be no doubt but that blood running down into the tracheobronchial tree is a thing that should be prevented so far as is possible. It not only is infected, but it furnishes a culture medium; it runs down into the tracheobronchial tree at practically every operation, and yet pulmonary abscess following tonsillectomy is exceedingly rare as compared to the number of tonsillectomies. One thing of importance in connection with the inspiration of blood is the fact that the cricopharyngeal pinchcock maintains the esophagus constantly closed except during swallowing. The larynx, on the contrary, is constantly open and it is located at the bottom of a funnel formed by the pharynx; into this funnel the blood pours from the wounds in the tonsils and nasopharynx. It is true that some blood is swallowed; but swallowing is intermittent, whereas the flow of blood into the larynx is constant.

3432 N. Broad Street.

CARCINOMA IN A LUNG ABSCESS CAVITY.

Dr. John D. Kernan and Dr. Arthur J. Cracovaner,
New York.

Chronic irritation and chronic inflammation have been reported and recognized as possible causes of, or possible sites for, the formation of carcinoma. In a review of literature we have been unable to find a report of the growth of carcinoma in a lung abscess cavity. We therefore feel that the following case should be reported:

H. W., a carpenter, aged 35 years at the time the diagnosis was made, was first admitted to the Lenox Hill Hospital on January 1, 1923, and gave the following history:

At this time, the patient's chief complaint was one of cough and expectoration of huge amounts of foul sputum. At 9 years of age, he had been ill for a long period of time with a condition diagnosed as bronchitis or bronchopneumonia. He had never been well since that time. Between the ages of 9 and 14 years, he was troubled with a nonproductive cough, and at the end of that period he began to expectorate a thin material which was not foul. The average amount of sputum brought up was about a pint a day. At 16 years of age, his tonsils were removed at the Presbyterian Hospital under local anesthesia. Two months later, he was admitted to the New York Hospital with a diagnosis of empyema of the left side of his chest. A rib resection was performed and the wound drained for about four months. Five months later, following a severe cold, he began to cough up foul smelling sputum. This cough and foul sputum persisted until his admission. In 1914, he was operated upon at New York Hospital for a lung abscess. The abscess drained for eight weeks. He improved markedly but still expectorated one cup of foul sputum a day. In August, 1922, he came under the care of Dr. Richard Jordan and the author at Lenox Hill Hospital.

At that time examination revealed a scar in the left posterior axillary line. Bronchoscopic examination showed a large amount of foul sputum coming from the left lower lobe bronchus. The diagnosis of lung abscess was made. There was no evidence of tuberculosis.

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In January, 1923, he was operated upon by Dr. Torek. A resection of the 9th and 10th ribs was performed and the lung abscess drained. A portion of the wall of the abscess cavity was excised. The bronchi were seen emptying pus into the abscess cavity. The biopsy was reported as inflammatory tissue from the wall of the lung abscess with marked hyperplasia of the bronchial epithelium. He was discharged from the hospital on May 8, 1923, with a bronchial fistula. A large clean cavity was still present. He showed no temperature, night sweats, cough or sputum. His general condition was fine and he had gained weight and was very active.

In November, 1927, a follow-up letter brought the following reply: "Patient has been in Ohio for three years. He is working and has married. The wound still drains a little. General condition much improved."

He was readmitted to the Lenox Hill Hospital on September 16, 1929. Up to one year before readmission he had been feeling very well. There was, up to that time, slight drainage from the wound which required one dressing a day. Lately, however, this drainage had greatly increased. He was also suffering from a shifting arthritis. He had occasional colds with some expectoration. About eight weeks before admission he noticed some blood on his dressing. This continued for two weeks, stopped for two weeks, and for the past four weeks had been occurring constantly. He had a cough and expectoration which had lasted for five weeks, but no loss of weight. Examination revealed an opening in the chest wall in the posterior axillary line at the level of the 9th and 10th ribs which was 2½ inches in diameter, and which exposed an irregular cavity about the size of an orange. The surface of the cavity was smooth except on the roof, where there was a 3-inch, irregular, cauliflower growth of tissue, part of which had a blood clot attached to it. Several attempts were made to control the bleeding by packing, but without success.

On October 17, 1929, the bleeding area was cauterized. Because of the hardness of the new growth tissue a biopsy was taken. Microscopic examination revealed the lung tissue to be replaced by a malignant new growth composed of nests and strands of pavement epithelium which was partly well differentiated and showed slight cronification and pearl formation. Other parts of the tumor showed varying degrees of anaplasia and many of the tumor cells showed mitotic figures. The stroma was, for the most part, fairly abundant and was rather dense and only moderately cellular. It showed con-

siderable inflammatory infiltration. Diagnosis was made of squamous cell epithelioma. Another biopsy, taken later, confirmed this diagnosis.

The patient was placed under the care of Dr. Pickhardt and was treated by insertions of Radon seeds and cauterization. In spite of this he developed extensive metastases, especially in the brain, and died several months later.

This, then, is a case of an abscess cavity which had been draining for about six years. A sanguinous discharge was the only symptom which called attention to the cavity again. Because of the hardness of the new growth tissue found in the cavity, and the irregular, bleeding, cauliflower nature of the growth, a biopsy was done which showed squamous cell epithelioma. In other words, it was a case of carcinoma forming in an old abscess cavity which was the site of a chronic inflammatory process.

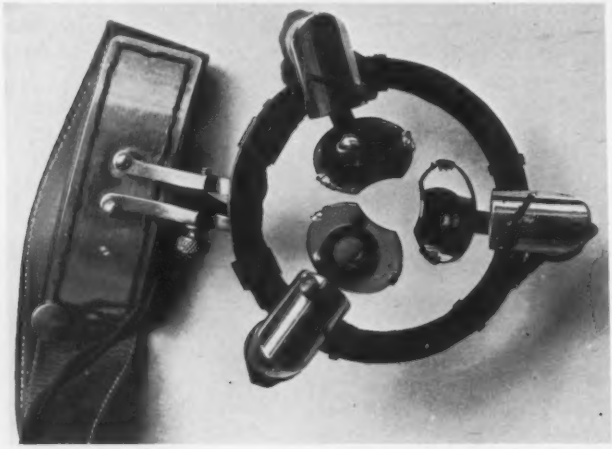
120 East 75th Street.

A NEW HEADLIGHT.

Dr. Duke R. Gaskin, Phoenix, Ariz.

As one glances through the current literature, he is sure to be impressed or rather depressed with the increasing number of new instruments which, with only a few exceptions, are modifications of older principles. Reflected light is certainly not a new principle.

I am submitting a new kind of headlight which I believe has advantages over the present group of electrically lighted headlights now being used by the profession.



Briefly summarizing, an operative headlight should be dependable, it should be comfortable both in regard to weight and amount of excess heat generated, it should furnish the required amount of illumination free from glare and color, and the rays should be parallel and located as near the central line of vision as possible.

The light is equipped with three six-volt bulbs under strong magnification. They are wired parallel so that the two remaining lights will function, should one bulb burn out. They are mounted around

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a small aluminum ring which works from a ball and socket connection, as does the ordinary head mirror.

The mirrors are located in such a way as to bring the rays of light in direct approximation with the central line of vision. They are plano in type, reflecting parallel rays as parallel rays. Using the three plano mirrors, we have avoided the use of the slight concave type of mirror which has heretofore been necessary in order to obliterate the shadow from the sight hole, but which converged the parallel rays, forcing the operator to work at or within the focal distance of the mirror. The magnifying lens are adjustble so that either parallel, convergent or divergent rays can be focused upon the mirrors.

The cone of light from the three mirrors fused at a distance of about nine inches and remain fused over a distance of about four feet. Therefore, with the light from the three mirrors superimposed upon the other, the total illumination is the sum total of the three six-volt bulbs under approximately a plus fifty diopter lens.

The light works from a transformer, and it is not necessary to work at full capacity, which diminishes the possibility of burning out the bulbs and allowing the light to remain perfectly cool.

922 Professional Building.

At the Annual Meeting of the American Laryngological, Rhinological and Otological Society held in Chicago, June 8-10, 1933, Dr. Max A. Goldstein, of St. Louis, was awarded a special medal for outstanding work in the study and rehabilitation of the Deaf Child. In addition to the special service for which this award was made it also included recognition of Dr. Goldstein's recently published book, "Problems of the Deaf." The award was made by Dr. Joseph C. Beck, President, speaking for the Council, at the Annual Banquet of the Society, Friday evening, June 9, 1933.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

April 17, 1933.

Dr. W. W. Wilkerson, Jr., Vice-Chairman, Presided.

Growth on Palate. Dr. W. G. Kennon.

Dr. W. G. Kennon reported a case in which a growth had been removed from the hard palate of a patient aged 67 years. The only symptom had been that the presence of the growth prevented the patient from wearing her dental plate. The tumor was a hard flat pedunculated one with its longer diameter running anteroposteriorly. Under novocain anesthesia it had been removed with a tonsil snare. The bleeding surface being cauterized with the diathermy coagulating current. The pathological report was as follows: "Tissue surrounded by a well developed but inactive stratified squamous epithelial layer. The central core is essentially fibrous. Some myomatous changes found. There are no indications of a malignancy."

Acute Thyroiditis. Dr. W. G. Kennon.

Dr. W. G. Kennon reported a case of acute thyroiditis which had developed during hospitalization of a patient for peritonsillar abscess.

Five days following the incision of the abscess the patient had complained of pain on the outside of the throat and of tenderness on pressure below the thyroid cartilage. She was only comfortable when the neck was flexed so that the chin rested on the chest. There was pain in the lower anterior part of the neck on swallowing.

The temperature ranged from 100° to 102.° W.B.C., 16,800. The thyroid gland became full and was definitely tender on palpation. At no time was there any disturbance of heart rate.

Mastoiditis With Some Interesting Features. Dr. W. G. Kennon.

Dr. Kennon reported an interesting case of mastoiditis which had occurred in a child 26 months of age following a severe attack of scarlet fever. Bilateral myringotomy was done for acute otitis media; two days following this there was swelling behind the left ear, and five days following there was subperiosteal abscess and mastoiditis were present bilaterally.

Mastoidectomies were performed at which time no corticle perforation was demonstrable. It was noted that the mastoid process was of adult development and each cell filled with liquid pus. The dura showed no pathology on either side.

The day following the operation the temperature rose to 105°. A slight swelling developed below the angle of the left mandible. This swelling had occurred on the second day postoperatively and the child was extremely ill. Blood transfusion was given. Thinking the swelling was due to erysipelas on the fourth day postoperative, the patient was given erysipelas antitoxin.

On the seventh day postoperative a discharge was discovered entering the left external auditory canal. This evidently had its origin in a parotid abscess which ruptured in to the canal.

The left wound showed no tendency towards healing; the right was healing very slowly.

On the eighteenth day postoperative the left lateral sinus was exposed. The sinus wall did not appear healthy, but the sinus was apparently filled with

blood. Thinking that it was jugular phlebitis or thrombosis, the left jugular vein was exposed. The vein appeared normal; however, several enlarged lymph nodes were found lying along its course. The wound was ligated in the usual manner.

Following the last procedure the temperature which had previously been of a septic type subsided by lysis, reaching the normal on the fourth day.

Drainage from the middle ear continued over a period of several weeks and from time to time small pieces of bone were removed.

The right mastoid bone healed after four months and has remained healed, although the patient has occasionally developed acute otitis media with discharge on this side. The left wound had never healed and by probing bare bone could be felt. Discharge continued from the left middle ear for two years.

A second operation was performed at the end of this period and several small sequestra were found over the lateral sinus. Two large fragments of sequestrated bone were removed. These were apparently the remains of the posterior canal wall. When the larger fragment was extracted the incus, eroded, could be seen lying loose in the middle ear. The long process of the malleus was practically gone. Healing was not complete at the time of this report.

Dr. Kennon summarized the unusual features of this case as follows:

1. The extraordinary rapidity of development of the mastoiditis.

2. The development of bilateral subperiosteal mastoid abscesses without demonstrable cortical perforation.

3. The extraordinary pneumatic development of the mastoid process in such a young subject.

4. The absolute absence of any inflammatory reaction in the mucoperiosteal lining of the mastoid cells.

5. The development of a paroid abscess with rupture through the fissure of Santorini.

6. The probable jugular phlebitis of lymph adenogenous or parotid origin.

7. The necrosis and sequestration of portions of the inner plate of the skull and of the bony posterior canal wall.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION ON OTO-LARYNGOLOGY.

Meeting of January 13, 1933.

Dr. H. J. Rothschild, President, presiding.

Oto-Laryngologic Practice from the Standpoint of Histologic Immunology.
Dr. Frank J. Novak, Jr.

Dr. Novak was a guest of the Academy and by invitation read a paper entitled: "Oto-Laryngologic Practice from the Standpoint of Histologic Immunology."

ABSTRACT.

The human organism is endowed with a mechanism of defense against pathogenic bacteria which is adequate within limits to protect it from invasion, or, failing to do that, to destroy the invaders. This mechanism of defense may be latent. It may then be stimulated or activated by means which are at hand. What is the nature of this mechanism, and by what means may it be controlled?

The infection of tissue by bacteria is followed by the formation of specific antibodies in the blood serum. There is also a synchronous mobilization of cellular elements. These elements are the polymorphonuclear leukocytes, large lymphocytes and monocytes. They are the microphages. Another group of cells, the macrophages, belong to the fixed cell type, but they possess the property of detaching themselves from the tissues of which they ordinarily form an integral part, acquire motility and wander great distances. They are attracted to the site of infection and, arriving there, exhibit their peculiar property of phagocytosis.

The sensitization of these cells—their activation—may be brought about by the injection of antigens, such as non-specific proteins, vaccines, filtrates, filtrates combined with vaccines.

It seems that intradermal injection of the antigen is a more efficient procedure than the subcutaneous injection. The antigens (filtrates) may be used on the skin as wet dressings, or by application to mucous membranes.

There has been an over-emphasis among oto-laryngologists upon the search for some ideal antiseptic to control infection of the ear, nose and throat. More attention should be directed to the mechanism of defense already present.

DISCUSSION.

Dr. Novak said, in response to Dr. Morton's query about diet, that he believed the recent emphasis upon diet in relation to infection is an important one. It is known that certain drugs have a measurable influence upon the immune reactions of the body. Aaron A. ———, in 1916-17, did some work in this regard. As to allergy, Pilot is convinced that arthritis following streptococcic throat infection is an allergic reaction. The therapy of such a reaction is a desensitization therapy. Pilot prepares a vaccine and then uses it in extreme dilution to start with. He injects it into the skin and proceeds gradually to desensitize the individual from the allergic standpoint. And the results are encouraging.

DR. J. S. REYNOLDS asked Dr. Novak, in the use of either stock or autogenous polyvalent vaccines, if malaise and a febrile reaction follows, what effect does that have on the results obtained.

DR. NOVAK believed that this reaction in itself is not an unfavorable one. He was of the opinion that most of the heat therapy at the present time in syphilitic involvements of the nervous system, the use of malaria to produce a febrile reaction, is in the nature of promoting an immune reaction. In using vaccines, Dr. Novak advocates a dosage less than that which will produce a febrile reaction. The immunological result is as great from the small dose which does not produce general symptoms.

DR. GEORGE C. DITTMAN said Dr. Novak mentioned the fact that a maximum of 1 cc. of the vaccine should be reached, and he wanted to ask Dr. Novak what amount he began the injections with and how many he used before arriving at that maximum? Also, does Dr. Novak use this in chronic sinus infection? Dr. Dittman said his experience in the use of vaccines in chronic infection had been rather unfortunate. He would also like to know what reaction, if any, is obtained should it be injected into a vein? And do the serums that have been put out recently, known as bacteriaphages, have any relation to this subject?

DR. NOVAK replied that very roughly the average cubic centimeter of vaccine (mono-vaccines, containing only one vaccine) contains about half a billion organisms. A combined vaccine will contain about a billion and a half. Why choose one cubic centimeter? As a matter of experience, it has been found that such a concentration and using no more than 1/20 of a cubic centimeter to start with will not produce an unfavorable response. If it is excessive, the dose is repeated and if there is no reaction from that the following doses are in proportion. Why choose the one cubic centimeter as the largest amount used? For the simple reason that it is difficult to inject more than 1 cc. into the skin unless one injects it in two or three places. There is no objection to increasing that dosage up to 2 cc. Dr. Novak called attention to one interesting fact in this regard, which is generally known, and that is, in an infection which is acute, i.e., characterized by high fever and great malaise, one is able to inject 1 cc. as a first dose without producing the usual local reaction. In a case of erysipelas complicating mastoiditis, if 1 cc. of streptococcus is injected, the patient does not get any worse, the temperature does not go any higher; on the contrary, the temperature tends to drop. It is an interesting, and to him unexplainable, fact that in these acute conditions a large dose of antigen may be given without producing aggravation of local or general symptoms. On the contrary, the general symptoms are alleviated. In regard to Dr. Dittman's question as to whether the vaccines are of value in chronic sinus infection, Dr. Novak said here again one presupposed that the necessary reconstructive surgery has been done—surgery aimed at producing suitable drainage. If, after this has been done, is there any role which various antigens can play to raise the resistance of that individual to enable him to overcome the sinus disease? Dr. Novak believed that everything he had said this evening points to this. One must make an effort to isolate the organism or organisms which are responsible for the pathologic conditions which exists. Having done that, the preparation of a suitable vaccine, the preparation of a filtrate is done, remembering that by injecting such an antigen a partial immunity is conferred on the part which is injected. The question was raised as to the effect of injecting vaccine into a blood vessel. In this connection, Dr. Novak cited Dr. Clausen's work at the University of Minnesota, of injecting the streptococcus vaccine directly into the blood stream.

In regard to the question of bacteriaphage, Dr. Novak said he had purposely avoided that subject this evening.

DR. KENNETH PHELPS asked Dr. Novak if he would discuss the treatment of common colds by vaccines, and the benefits which may be expected from various types of vaccines.

DR. NOVAK felt that one could conservatively and rationally put it this way. In the first place the etiology of the common cold is not known. It is known, however, that there is an associated series of organisms in the common cold in the upper respiratory passages. Dr. Novak believed that these organisms are the ones responsible for suppuration in the middle ear, and the sinus cases. He also believes that the use of immunologic measures prevents or mitigates

the complications. It is known that there are individuals who are prophylactically immunized to these organisms in the nose. Dr. Novak believed that some individuals can be solidly immunized against the common head cold, and, by the same token, he believed there are certain individuals who cannot be immunized at all.

Dr. V. J. Schwartz said he wished to add his expression of appreciation for Dr. Novak's very clear and concise presentation of a subject which is intrinsically complicated. He covered a large amount of ground in the time available. Dr. Schwartz said it seemed to him in the matter of therapy much, if not all, depends upon the isolation of the principal offending organism, and almost everything else hinges on this all-important matter. He wished to ask Dr. Novak how it is possible to isolate this bacterium, and when there are (as is almost always the case) two or three principal bacteria involved, how it is possible to tell which is the one which must be treated or the one against which major efforts are to be directed. When Dean first started his work on the treatment of sinuses in children, the original plan was to sterilize the interior of the nose with alcohol, using sterile trocars, of course, and washing the secretions of the sinuses into a sterile saline solution. From this, bacterial studies were to be made. Dr. Schwartz said this had always appeared to him as a most unsatisfactory procedure and far removed from the objective aimed at. He wondered what technic Dr. Novak uses for this purpose. His own impression was that if one can isolate the one or two principal bacteria involved, an important problem will be solved because a vaccine can readily be prepared against these.

Dr. Novak said Dr. Schwartz raised a very important question in regard to therapy and the isolation of the particular organism in a given case which is the offending organism. If one cultured the noses of various ones assembled here, there would be found various organisms no doubt. In the front portion of the nose, the staphylococcus; in the middle, the micrococcus, and in the back portion of the nose, the diphtheroids, etc. The tonsils and adenoids frequently contain the streptococcus. Let it be assumed that one has made a culture from the throat and finds three organisms. In order to determine which organism or organisms to include in the vaccine, two procedures are available. The offending organism may be identified serologically, or a *very dilute* suspension of killed culture of each of the organisms may be injected intradermally into the patient. The one producing a reaction is selected as the probable offender, and a vaccine prepared from it.

Dr. F. N. Knapp asked Dr. Novak if he could explain why it is that one year there is an epidemic of sore throats, and the next year possibly an epidemic of antrum trouble or some other infection in the head.

Dr. Novak replied that this involves the whole question of virulence, about which very little is known. To those particularly interested in the various aspects of this subject, Dr. Novak recommended the study of Hadley's work at the University of Michigan; also the work of Falk as to the "charge" which various bacteria carry. Falk suggests that when something affects a bacterium to increase the charge, that organism increases in virulence; and when it loses the charge, it loses its virulence.

Dr. Walter E. Camp said he was very much interested in Dr. Novak's description of local tissue immunity, and he wondered if Dr. Novak would tell when the infection reaction starts and about what time the maximum immunity develops? He also wished to ask whether the vaccines are not more valuable when used intravenously? Dr. Clausen, at the University of Minnesota, has been using them intravenously in rheumatism, and Dr. Camp wondered if this method of administration would enhance their value in building up the immunity factor.

Dr. Novak replied that, theoretically, the immune reaction starts almost synchronously with the injection of the antigen; it is a question of hours, not days, before the immune bodies are increased at the site of the lesion. Soundly conceived immunological efforts offer some measure of success. In regard

to intravenous administration, Dr. Novak stated he had already referred to the work of Dr. Clausen, pathologist at the University of Minnesota. As far as his own work is concerned, Dr. Novak said his experience with intravenous injection is nil; he had not used antibacterial serum, excepting antimeningococcic serum, or any other antigen by injecting into the blood stream.

DR. H. J. ROTHSCHILD said Dr. Novak brought up the question of filtrates and the inoculated filtrates, which are the bacteriaphages. He wanted to know if Dr. Novak had had any experience with the bacteriaphages put out by the larger laboratories, such as those of Lilly & Company, and, if so, with what results?

DR. NOVAK replied there are those who refer indiscriminately to filtrates as bacteriaphages, but he himself felt that a distinction must be made between them. A filtrate is not strictly a bacteriaphage, but it may become a bacteriaphage after it is prepared for that particular thing. Dr. Novak felt he was not competent to discuss the question of bacteriaphages. As to the clinical results produced by the phage, Dr. Novak said he did not know.

Dr. Novak said he wished to thank the members of the Academy for the invitation to address them and for the fine attention to the subject which he had been trying to cover in this length of time.

DR. ROTHSCHILD, on behalf of the members, thanked Dr. Novak for coming to this meeting, and said it had been a long time since this Society had had so pleasant and profitable an evening.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION OF OTO-LARYNGOLOGY.

Meeting of February 10, 1933.

Dr. Harold J. Rothschild, President, presiding.

DR. L. R. BOIES reported five cases of tumors of the nose and throat.

Tumors of the nose and throat are treated on the Oto-Laryngologic Service of the University Hospital, with the aid of Dr. Peyton, who is in charge of the work of the Cancer Institute, and Dr. Stenstrom, who directs the work in Physical Therapy. Approximately twenty-five nose and throat tumors are seen per year, exclusive of lesions of the esophagus or lungs.

Case 1. Carcinoma of the Larynx: This patient is a farmer, age 55 years, who was referred to us in September, 1932. Fifteen months previously he had experienced the gradual onset of hoarseness, for which he consulted a physician. A submucous resection was performed. About two months later the hoarseness had not improved and he consulted a second physician. The latter made a diagnosis of carcinoma and advised operation, which was declined. The patient was treated with radium (record of dosage was not obtained) and he was also given X-ray therapy. The examination on admission to the University Hospital revealed a malignant growth which apparently had originated on the right false vocal cord and was extending into the right pyriform sinus. He had a moderate amount of interference with motion of the right cord but no appreciable stridor. He had a slight interference with deglutition.

There were no palpable glands in the neck and he was in excellent general physical condition except for some prostatic disturbance.

Under local anesthesia a two-stage laryngectomy was performed; the second stage was completed on Oct. 15. Recovery was uneventful. At operation the tissue was excised so widely in the right pyriform sinus that it was impossible to get a tight closure of the floor of the larynx. The resulting fistula is now almost closed; it has been allowed to heal in gradually, aided by three minor plastic procedures. The nasal feeding tube has been in place almost continuously since the second stage because of this fistula. It has been removed at times and it was found he can swallow fairly well except for some loss through the fistula. He retains his weight and feels good in spite of the prolonged tube feeding. There is no evidence at this time of any recurrence. (It might be added here that he has had a prostatic punch operation in the interim since his laryngectomy.) X-ray therapy will be given as soon as the fistula is entirely closed.

Case 2. Carcinoma of the Antrum, Grade IV: This man, who is now 51 years old, first came here on April 14, 1931. Three years previously he had had a swelling of the right side of his face. A tooth was pulled and there was a purulent discharge from the socket and also from the right nostril. The swelling subsided but later returned. The discharge had continued. In February, 1931, the right side of his hard palate began to bulge and he consulted a doctor who referred him to this hospital. The examination on admission revealed a firm, swollen right cheek. Tumor tissue protruded through the hard palate into the mouth.

On March 17, 1931, the right half of his hard palate was removed with diathermy current after the ligation of his right external carotid artery. The malignant tissue filled the antrum, the posterior ethmoid area and had broken through posteriorly into the lateral portion of the epipharynx. All tumor tissue was removed. The lining of the operative cavity was coagulated and four radium bombs were packed into the cavity to give him 3500 mgm. hours of filtered radiation. Recovery was uneventful. A course of X-ray therapy was administered before the patient left the hospital. About two months later some swelling developed in the right zygomatic area and additional X-ray therapy was given to this area.

The operative cavity has healed and at this time is cleanly epithelized. From the roof of the cavity polypoid tissue has become evident from time to time. Numerous biopsies have been taken, but all have been negative. Two months ago a mold of dental stent containing 50 mgms. of radium was fitted into the operative cavity to radiate this polypoid area, and 200 mgm. hours were given. This treatment so far has controlled the polyp formation.

It is now almost two years since the radical removal of this Grade IV squamous celled carcinoma. A prosthetic device is to be made to bridge over the palate defect.

Case 3. Fibroma of the Nasopharynx: This 15-year-old boy was presented at the meeting of the Academy here a year ago. At that time he had had a large fibroma of the nasopharynx removed by the use of diathermy currents. The tumor had been present, according to the history, about two and one-half years and had its origin at the base of the sphenoid and in the sphenoethmoidal recess. The growth had practically occluded the nasopharynx, pushed the uvula and soft palate forward and, viewed through the oral cavity, hung as low as the base of the tongue. At the time of the original operation the tumor was removed almost up to its base and several radon seeds (totaling 900 mc. hours) were inserted. Considerable bleeding occurred and for this reason complete removal was not attempted.

Six months later the patient was again hospitalized and the base of the tumor attachment was coagulated. Three days later a serious hemorrhage occurred, necessitating a postnasal pack and subsequent transfusions.

Today unless you were told where the original attachment of the tumor had been you would have difficulty in locating the site.

Case 4. Carcinoma of the Tonsil: This patient, 51 years old, experienced a sore throat and spat up some blood in March, 1931. Gradually he became

unable to open his jaws widely. The left side near his last molar tooth seemed to be involved in a "sore" for which he had some dental attention. About one year later he was seen at Ancker Hospital in St. Paul. A biopsy was taken and he was given some X-ray treatment. Two months later he was seen in the Tumor Clinic here. At that time our examination revealed a tumor mass which seemed to have originated in the left tonsil, had extended into the adjacent margin of the left upper alveolus and soft palate and down the lateral pharyngeal wall almost to the pyriform sinus, and had just broken through the left cheek. Biopsy revealed a squamous celled carcinoma, Grade IV.

On Aug. 29, 1932, under avertin anesthesia supplemented with chloroform, the patient was placed on a suspension laryngoscope and this extensive growth was planted with seeds of radium emanation totaling 2640 ms. hours. His jaw was rather firmly ankylosed and the left cervical area indurated, but no discrete glands were detected. The area of induration was included in the distribution of the seeds.

The interesting feature of this case is the fact that this patient today exhibits no definite sign of his malignant growth, experiences no real discomfort (he still has the ankylosis) and has no sense of ill-being. There iis a firm fibrous scar at the original site of the main tumor mass in the region of the left tonsillar fossa.

It is not expected that this is a cure; the tumor has been checked, however, and in the five months since the radium implantation it has shown a surprising response to radiation.

Case 5. Cancer of the Epiglottis: This 75-year-old woman, over three and one-half years ago, experienced a sense of dryness and irritation in her throat. This gradually increased to a sense of fullness or lump, aggravated by swallowing. Eventually she reduced her diet to semi-solids and liquids. She lives on a farm and, having always enjoyed excellent health, did not seek any medical advice up to six months before she was first seen here. Twice within this six-month period she had had tonsillectomy advised to relieve her distress.

In May, 1932, she was examined in the dispensary and a malignant growth was noted which apparently had originated on the epiglottis and which now involved the pyriform smus and had extended into the larynx. She was admitted to the University Hospital for further study, where it was found she had an advanced hypertensive heart condition.

On May 21, 1932, thel larynx was implanted (after preliminary tracheotomy) with twenty-five seeds, giving her a total of 3590 mc. hours of emanation—a tremendous dose. Avertin supplemented with chloroform inhalation was used for the anesthetic. This was followed by a course of X-ray. She returned to her home with no particular discomfort except that caused by the tracheotomy tube.

About six weeks after the radium implantation she returned to the Tumor Clinic for follow-up. The day of her return she coughed out the tracheotomy tube. Examination of her larynx showed a surprising regression of the tumor tissue. A moderate amount of edema of the epiglottis and the side of the larynx was the only suggestion of pathology. She experienced no difficulty in breathing, and the tracheotomy tube was not replaced.

On Oct. 15, 1932, some glands in the left side of her neck were implanted with seeds (nine implants—1530 mc. hours) after exposing them by incision.

The interesting features of this case are: (1) The long history of trouble: (2) marked regression of the growth from the radium; and (3) lack of discomfort nine months after this treatment, which is not expected to be curative.

Petrositis. Dr. Nelson Youngs (by invitation).

Dr. N. Youngs reported the following case of petrositis.

The patient, R. L., No. 612136, age 10 years, was admitted to the University Hospital Dec. 4, 1932, and died Dec. 26, 1932.

Six weeks before admission the patient had developed bilateral otitis media. Two days later his attending physician did a bilateral myringotomy. The right ear discharged a small amount of thin serous discharge for a few days, while the left ear discharged a large amount of pus until four weeks before admission to the University Hospital. Because of general malaise and septic temperature (104° to 104.5°) the patient was confined to his bed from Oct. 23rd until Nov. 15th. After that he felt better and returned to school on Nov. 21st, 22nd and 23rd. On Nov. 23rd (11 days before admission), he came home from school complaining of left frontal headache, photophobia and diplopia. These complaints remained about the same and on Dec. 3rd and 4th, he had projectile vomiting and on admission he complained of pain in the left eyeball, photophobia and diplopia.

General examination was essentially negative. The pupils were equal and reacted to light and accommodation. The media and fundus were negative. There was paralysis of the left external rectus. The right ear was negative. On the left there was slight mastoid tenderness, the canal was dry and the drumhead thickened.

Rinne: right, +; left, —. Weber: right, <. Whispered voice: right, 15′; left, 5′. Watch: right, 3 1/3′; left, 1 1/3′.

Neurological examination showed paralysis of the left lateral rectus, diplopia, a negative Kernig. There was no reck rigidity.

On Dec. 4, 1932, at 11:30 p. m., an exploratory mastoidectomy was done. The soft parts were normal; small celled sclerotic type of mastoid and the cells were filled with swelled mucosae. The dura was exposed near the antrum. On Dec. 5th, pus from the mastoid showed staphylococcus and Gram negative rods. On Dec. 15th, the spinal fluid cultures yielded Gram negative rods.

Laboratory Data: Dec. 4, 1932: Hb., 83; W.B.C., 21,400; spinal fluid pressure, 150; cell count, 20.

Dec. 5, 1932: W.B.C., 26,500.

Dec. 19, 1932: Hb., 63; W.B.C., 19,950; spinal fluid pressure, 100; cell count, 49.

Dec. 21, 1932: Hb., 59; W.B.C., 16,950; spinal fluid pressure, 50; cell count, 1750.

Dec. 22, 1932: Hb., 67; W.B.C., 17,400; spinal fluid pressure, 100; cell count, 190.

Postmortem showed basilar meningitis and osteomyelitis of the petrous tip with erosion into the posterior fossae.

DISCUSSION.

Dr. Horace Newhart said the outstanding lesson from this case is the need of earlier surgical intervention. Operation should not be delayed after there is reason to suspect a possible impending endocranial complication, as was indicated in this case by the abducens involvement. Often one observes in cases of endocranial complication a remission of symptoms following operation, which is misleading and gives a false sense of security, as occurred in the case reported by Dr. Youngs. From the findings at autopsy it is probable that nothing could have been done after admission that would have changed the outcome. It is well known that a large percentage of cases with the Gradenigo syndrome recover if operated early. However, the Gradenigo syndrome is by no means synonymous with osteitis of the petrous tip.

General Septicemia Following Mastoiditis with Bezold's Abscess. Dr. H. E. Clark.

Dr. H. E. Clark reported the following case of general septicemia following mastoiditis with Bezold's abscess:

The patient, C. P., No. 612698, age 49 years, was admitted to the University Hospital, Dec. 20, 1932; discharged Dec. 29, 1932; re-admitted on the 30th of December, and died on the 13th of January, 1933.

He gave a history of intermittent pain in the right ear for the three months previous; pain, tenderness and swelling over the right mastoid for the past three weeks. There was no history of nausea, vomiting, headache or vertigo. His past history was negative with regard to his ears.

General physical examination was negative. Examination of the right ear showed the auricle displaced anteriorly. There was pain, tenderness and fluctuation over the right mastoid and drooping of the posterior superior canal wall. There was no discharge; the membrane was thick and no perforation could be seen. The tip could not be felt and a diagnosis of Bezold's abscess was made. On the left side the drumhead was negative and there was a healed perforation.

Rinne: right, +; left, +. Weber: right, <. Schwabach: right, N.; left, N. Whisper: right, 4"; left, 15'. 3-A: right, 25; left, 5. 2-A: right, 25; left 5.

There was no nystagmus, and neurological examination was negative.

A diagnosis was made of acute mastoiditis right, subperiosteal abscess, Bezold's abscess.

A simple mastoidectomy was performed the day after admission, Dec. 21st. On going through the periosteum at the inferior angle of the incision, pus welled up from the tissues of the neck. The mastoid was of the large celled type, broken down and filled with pus. A fistula was demonstrated from the tip into the tissues of the neck. The dura was not exposed.

For nine days postoperative the patient's temperature was either normal or subnormal. He sat up on the third postoperative day and was out of bed on the fifth, feeling fine. He was discharged nine days after operation and instructed to return to the outpatient clinic daily for dressings. On the following day (tenth postoperative) he reported at the dispensary complaining of headache, gastric distress and constipation. His temperature was 103.8°, and he was re-admitted to the hospital. At this time the physical examination was essentially negative. The pupils reacted to light and accommodation, and the ophthalmoscopic examination was negative except the veins were slightly dilated in the nasal portion and both discs were indistinct. The neurological examination was negative.

On the following day he had a septic temperature and a feeling of chilliness. Blood cultures were made which showed pneumococcus type III. Spinal puncture showed the fluid to be clear, pressure 100, and 1 cell present. On Jan. 2, 1933, he continued to run a septic temperature, with no objective or subjective findings except horizontal nystagmus on looking to the left. The patient had previously been constipated, but on Jan. 9th he developed diarrhea and abdominal tenderness. There were no other subjective or objective findings, however. A consultation was held with the surgical staff. There was tenderness over the abdomen, more marked in the lower left quadrant, some voluntary rigidity and moderate distension. It was felt that he had a peritonitis.

A diagnosis was made of probable sinus thrombosis on the basis of positive blood cultures and no other pathology to explain the findings.

Two days later (Jan. 11th), the internal jugular was exposed and ligated. The lateral sinus was exposed, which appeared normal. It was incised, but no thrombosis was found. It bled freely from both the superior and inferior ends.

The patient died on Jan. 13th; temperature, 107°.

Autopsy findings were entirely negative except for the postoperative findings. Final diagnosis: Septicemia—pneumococcus type III.

Laboratory Chart: Dec. 20, 1932: Hgb., 90.

Dec. 21, 1932: W.B.C., 9,150; Hgb., 87.

Dec. 30, 1932: Mastoid cultures, pneumococcus; W.B.C., 9,050; Hgb., 85.

Dec. 31, 1932: Blood cultures taken; spinal fluid, clear; pressure, 100; cells, 1; Queckenstedt: right, 200; left, 200.

Jan. 5, 1933: R.B.C., 4,250,000; W.B.C., 13,500; Hgb., 75.

Jan. 7, 1933: Blood cultures, taken; W.B.C., 9,800; Hgb., 82; spinal fluid, clear; pressure, 145; cells, 1; Queckenstedt: right, 230; left, 155.

Jan. 9, 1933: Blood cultures, pneumo. III; W.B.C., 15,400; Hgb., 84.

Jan. 11, 1933: W.B.C., 13,800; Hgb., 80.

DISCUSSION.

DR. HORACE NEWHART said this is another one of those cases in which the results probably would have been different had the patient been operated earlier.

Meningitis of Otogenous Origin, with Recovery. Dr. Horace Newhart.

Dr. Horace Newhart reported the following case of meningitis of otogenous origin, with recovery:

R. S., male, age 20 years, was admitted to the University Hospital late in the evening of Jan. 17, 1933. The patient had had a severe attack of influenza beginning about Dec. 24th, and he had lost about thirty pounds in weight. His only ear symptom was tinnitus in the left ear. On Jan. 13th (four days before admission), he had pain in the left ear, following in two days by nausea, vomiting, headache, chills and fever. A paracentesis had been performed the day before admission, without discharge from the middle ear. The right mastoid had been operated several years ago.

Examination showed a young man of slight build, considerably emaciated, suffering great pain behind the left ear, and with rigidity of the neck. The pupils were equal and reacted normally to light and accommodation. A slight paresis of the right external rectus was present and a questionable exophthalmos of the right eye. There was noted a slight horizontal and rotary nystagmus on looking to the left. The right postauricular region showed an old mastoidectomy scar. The left canal was filled with dry blood but no purulent discharge. There was notable tenderness at the root of the zygoma and over the mastoid region. Hearing for the right ear was slightly decreased; for the left, greatly diminished. The Weber lateralized to the left. The Rinne was positive for both ears. The nose and throat were negative except for a deviation of the septum and abundant purulent postnasal discharge. The neurological examination was negative except for the questionable paresis of the right external rectus, and possibly a slight Kernig. Cerebration was slow. The great distress caused by movement of the neck made it difficult to obtain Roentgenograms, but those obtained showed both mastoids were located well forward.

Laboratory Report: Spinal fluid very cloudy and under a pressure of 300 millimeters, with a cell count of 1845; leukocyte count, 14,700; hemoglobin, 84.

Spinal fluid and blood cultures were later reported negative. Pus from the perisinus abscess contained streptococci and pneumococci.

After consulation with Dr. J. C. McKinley, of the Department of Neurology, a diagnosis was made of circumscribed purulent meningitis, with a very doubtful prognosis. It was decided to operate immediately. The soft parts, except for moderate edema over the zygomatic root, were normal; the mastoid process was under-developed, and the tip cells were very small and filled with thickened membrane and a slight hemorrhagic exudate. The mastoid body was largely sclerotic. The lateral sinus was far anterior, covered with necrotic bone. A perisinus abscess was found, containing about 3 cc. of thick yellow pus under pressure. The sinus wall was covered with granulations. The adjacent bone was very sclerotic and the antrum was not exposed.

Because of the patient's condition the operation was terminated to avoid further concussion, in the hope that the thorough drainage of the perisinus abscess would yield good results.

The postoperative diagnosis was circumscribed purulent meningitis from a perisinus abscess without definite symptoms of a preceding otitis media.

The patient made a rapid and uneventful recovery except for a moderate swelling and tenderness over the zygoma and mastoid tip.

A rare and interesting phenomenon was noted in this case, in that for three days after operation moderate pressure over the zygomatic region caused a

definite movement of the auricle upwards and forward, whereas postauricular pressure produced a distinct auricular movement upward and backward.

In our opinion, prompt surgical intervention alone was responsible for recovery in this case.

DISCUSSION.

DR. V. J. SCHWARTZ asked if the patient had any intraspinal treatment.

Dr. Newhart said he had had none except for two spinal punctures for culture, on Jan. 20and 21, which showed clear fluid, the first yielding 300 cells and pressure of 200 mm., and the second normal as to cells and pressure of 190.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION ON OTO-LARYNGOLOGY.

Meeting of March 10, 1933.

Dr. H. J. Rothschild, President, presiding.

Case 1: Parotid Fistulae Following Mastoidectomy. Case 2: Papilloma of the Tongue of a Child. Dr. John J. Hochfilzer.

Dr. Hochfilzer reported two cases.

Case 1: Parotid fistulae following mastoidectomy.

The patient, a girl 15 years of age, was admitted to the Ancker Hospital eight years ago with scarlet fever. Soon after admission she developed an acute otitis media on the right side, which terminated in an acute mastoiditis, for which she was operated and made an uneventful recovery. The discharge from the mastoid wound had practically cleared up at the date of her discharge, on Jan. 23, 1925.

However, this slight discharge from the wound never ceased entirely, and the patient noticed a constant seeping of clear, watery fluid out of a small opening at the lower end of the wound. This persisted for seven years, during which time an operation of the fistula was performed but with no success. The patient had no discomfort from this condition save a constant damp feeling behind her right ear. She noticed the discharge increased when she ate certain foods of a tart nature, such as pickles and peanuts.

The physical examination revealed nothing abnormal in her ears, nose or throat except the fistula. Her hearing function was within normal limits. Examination revealed two postoperative scars directly behind the right auricle. At the lower end of the first operative scar is an indentation the size of a pinhead, but no opening is visible. Several attempts to probe the fistula were unsuccessful. Upon giving the patient peanuts to eat, the flow from the fistula immediately increased so that 10 cc. of fluid could be collected in about 10 minutes. The chemical analysis of this showed the presence of ptylin, the ferment of the parotid gland.

There are several factors which may be responsible for a fistula of this sort: (1) An abnormally large parotid gland extending backward over the mastoid process. (2) A previous inflammatory process may have increased the size

of the parotid gland and pushed it back into the operative field. (3) A very marked periostitis or Bezold abscess of the mastoid process which distorts the topical anatomical relations and thus at operation may lead to an incision which is injurious to the parotid gland.

Various methods of treatment, none of which is 100 per cent sure, and all failing in some cases, have been advised for parotid fistula, namely: (1) Chemical cauterization, using silver nitrate of chromic acid; (2) Galvanocaustic; (3) Excision of the fistula and plastic surgery.

In the case just reported, Dr. Hochfilzer used electrocoagulation. A very fine needle, such as is used for epilatory purposes, was inserted into the fistula and the area of the suspected tract cauterized. The discharge decreased shortly thereafter and stopped entirely one week after treatment was instituted. Of course, the facial nerve was watched very closely during the administration of the treatment.

Case 2: Papilloma of the tongue of a child. The patient, a boy 14 years old, was brought to the Wilder Dispensary because of the condition of his tongue. The mother stated he was born with this growth in his mouth, but that lately it had increased considerably in size, which fact worried her. The boy had no discomfort whatever from the condition except that it would start to bleed when he masticated his food.

Examination of his mouth showed a large cauliflower-like sessile tumor, of a pinkish color, on the right side of the tongue, starting at the middle third and involving the marginal region and base and the plica triangularis, then continuing to the anterior faucial pillar, the upper tonsillar pole and the right portion of the uvula. There was no gland enlargement. The biopsy report was "papillomatous growth."

Two facts are interesting in this case: (1) We are dealing with a congenital form of papilloma if the statement of the mother is correct. In a recent article dealing with this subject, New reports the age of sixteen as the youngest person in his series afflicted with this disease. (2) The extensive involvement of structures by the growth.

The treatment in this case would undoubtedly be diathermy. However, the mother refused to have anything done for the boy.

BOOK REVIEW.

Problems of the Deaf. By Max A. Goldstein, M.D., F.A.C.S.; Director, Central Institute for the Deaf; Professor of Otology, St. Louis University, 1900-1912; President, American Academy of Ophthalmology and Otolaryngology, 1902; President, American Otological Society, 1928; President, American Laryngological, Rhinological and Otological Society, 1931; President, Society of Progressive Oral Advocates, 1917-1933; Editor, THE LARYNGOSCOPE; Editor, ORALISM AND AURALISM. 580 pages; 273 illustrations; The Laryngoscope Press, St. Louis, 1933. Price $6.00.

It is timely that an eminent authority like Dr. Goldstein should come out with a volume which will forever remain as a masterpiece in otological literature and which will prove of invaluable assistance to all who are interested in the problem of the deaf or deafened.

In the preface Dr. Goldstein states that the study of the deaf child must be regarded not only as a clinical entity but also as a vital factor in the educational and economic life of the community. It requires more than a knowledge of clinical pathology and surgery to minister to his needs and to intelligently advise the parents. The schoolroom and the special problems of the qualified teacher, the preparations for his social contacts and the adjustment of his handicaps are as much a clinical amphitheatre as is the hospital or the bedside.

It is indeed unfortunate that it has taken over forty years of Dr. Goldstein's experiences to prove to the otologists and practitioners at large that something of real value can be done for those who have been unfortunate enough to lose their hearing. Dr. Goldstein, in his modesty, but barely touches on the great work which he is doing but his book is a vast monument to him personally and to the cause of the Handicapped Child.

The volume is divided into nineteen chapters, each one of which takes up a definite problem concretely. One is particularly impressed with certain chapters which are of special value because the information therein contained can be found elsewhere only with difficulty. Of great importance are the chapters on the training of teachers, the hard-of-hearing child, the physician and the deaf child, the parent of the deaf child, and schools for the deaf. In the latter chapter is a definite compilation of interesting statistics of each school with the State laws governing them.

The book is profusely illustrated with excellent line drawings and halftones which are sufficiently simple for one to understand at first glance.

This book, "Problems of the Deaf," will be of two-fold value. In the first place it will serve as a guide to parents, teachers and physicians who wish to know more fully how to take care of the young child, particularly one of pre-school age who shows a definite retardation in hearing. Secondly, it will stand for all times as a reference book to which one can refer at any moment and feel that he has the knowledge of a real authority for any problem along these lines that he wishes to solve. Considering its value, it is truly to be hoped that this volume will find its way into the hands of the thousands of individuals who wish to know the last word about the care of the deaf or deafened child or adult.

H. H.

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